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VOLUME 46 NUMBER 2

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AUGUST 1947

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO 10, ILLINOIS. ANNUAL SUBSCRIPTION, $6.00

Entered as Second Class Matter Jan. 14, 1925, at the Postoffice at Chicago, Under the Act of Congress of March 3, 1879

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MANAGEMENT OF RESPIRATORY COMPLICATIONS OF POLIOMYELITIS

THOMAS C. GALLOWAY, M.D.
EVANSTON, ILL.

THE PROBLEM of severe bulbar poliomyelitis is largely one of secretional obstruction of the respiratory tract, with secondary anoxia. The satisfactory result of management based on that idea and the rapid change in the aspect of apparently hopeless cases on clearing the airway suggest that anoxia is more important than the virus infection itself.

Poliomyelitis occurs chiefly in sporadic epidemics, and the physician who meets it for the first time is likely to find the respiratory complications bewildering. In this discussion, based on four years' experience of poliomyelitis and previous experience of respiratory obstruction, an attempt will be made to visualize the mechanisms involved and to present in some detail the measures that will give relief. The prevention and the relief of anoxia are the urgent indications.

The statistics accumulated by Priest, Boies and Goltz [1] in the course of their fine management of a large series of cases indicate that the principles expressed in 1943 [2] are correct.

Respiratory complications have been described as of three types: (1) those due to spinal paralysis involving the diaphragm or intercostal muscles or both; (2) those due to bulbar paralysis involving the mechanism of deglutition; (3) those due to disturbances of the respiratory center.

The first type may be uncomplicated by any involvement of the central nervous system. They usually respond excellently to the use of the respirator, the breathing falling into a good rhythm, with relief of symptoms and usually fairly rapid convalescence and return of function. Here secretions rarely bother, but cough and tussive squeeze are lost, so that bronchial secretion may accumulate and secondary

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Read before the Illinois Medical Society, May 13, 1947.

From Northwestern University Medical School and Evanston Hospital.

1. Priest, R. E.; Boies, L. R., and Goltz, N. F.: Tracheotomy in Bulbar Poliomyelitis, Tr. Am. Laryng., Rhin. & Otol. Soc., 1947, to be published.

2. Galloway, T. C.: Tracheotomy in Bulbar Poliomyelitis, J.A.M.A. **123**: 1096 (Dec.) 1943.

infection ensue. Ciliary action and air movement usually keep the airway clear, especially if postural drainage is employed.

Disturbances of the second type, with bulbar paralysis, are most important and demand recognition of the various factors involved. I have described a typical case in which unswallowed secretions filled the larynx, the upper part of the trachea, the pharynx and even escaped from the mouth and the nares. The patient had become depressed, restless, irrational and disoriented and showed marked respiratory arrhythmia. The condition had been ascribed to the virus infection. When the patient became cyanotic and comatose, a laryngologist was called. Respiration ceased for about three minutes, but after tracheotomy, aspiration and artificial respiration the patient was placed in a respirator, and consciousness, good color and good pulse were restored in forty-five minutes. It is hard to believe that this depression so quickly relieved could be due to anything but anoxia.

Caution has been urged in using the respirator in such cases. It has been my experience that if the airway is cleared of secretion or a by-pass is provided by tracheotomy, the patient does unusually well with the machine. It should be realized that if voluntary or artificial respiratory movements are made while the upper air passages are filled with more or less viscid secretion, this will be drawn into the lung bed, virtually drowning the patient. Since this seems hard to visualize, a simple model was made to demonstrate the mechanism, which is shown in figure 1.

Disturbances of a third type, due to involvement of the respiratory center, probably can occur, but the picture is confusing. Arrhythmia and incoordinations are considered typical. One often sees a patient with shallow, interrupted, cogwheel and wavelike respiratory movements. At times it almost appears as if the intercostal muscles and the diaphragm were opposing each other. I have seen such arrhythmia controlled for a time by sodium pentothal, which would hardly be the case if the cause were central depression. Likewise, these movements often quickly disappear on clearing the airway, especially after tracheotomy, with fairly normal rhythm and movement being restored. A simpler explanation of this type of respiration is that reason in the conscious patient, instinct in the depressed patient and reflex in the unconscious patient keep them from drowning in their own secretions, as they would do if they breathed deeply and normally.

Of course, all combinations can occur, and it is probable that overwhelming purely central infections do occur. However, the effects of virus infection with edema are extremely difficult to separate from those of anoxia even by histologic examination, as shown by Courville [3]

3. Courville, C. B.: Pathology of the Central Nervous System, Mountain View, Calif., Pacific Press Publishing Association, 1945.

and others. From a number of cases of apparently overwhelming infection in which I have seen dramatic relief after establishing and clearing the airway, I am persuaded that anoxia is much more often the factor than severe infection. At any rate, this assumption offers

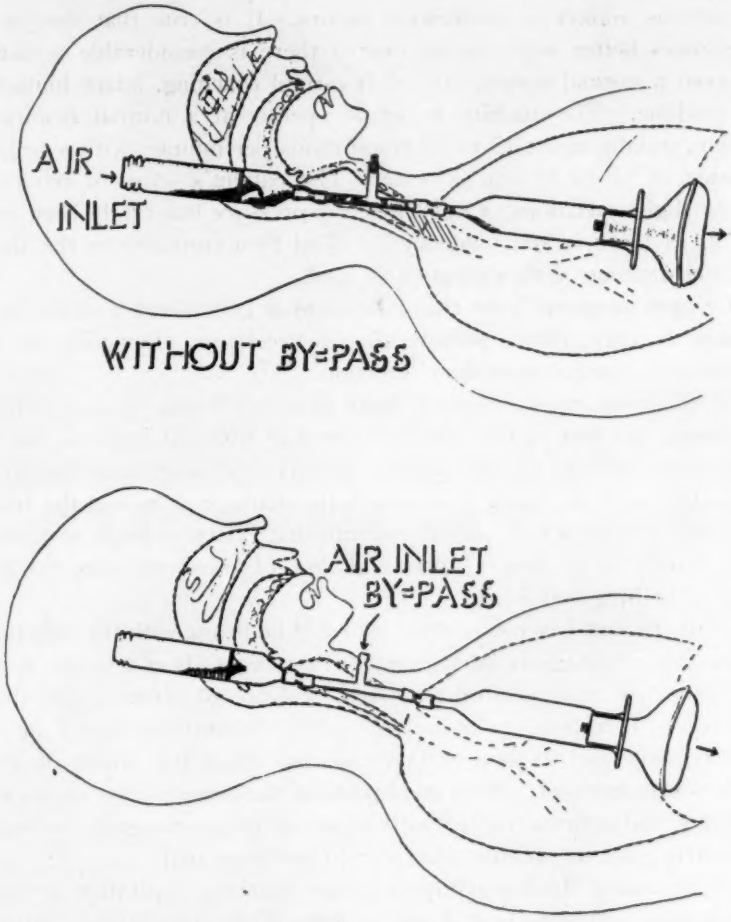

Fig. 1.—A model to simulate the effect of voluntary breathing or a respirator when there is fluid in the upper airway. When the by-pass is open, no fluid is drawn in. With moderately viscid fluid, intravenous tube, connector and asepto-syringe it acts as shown.

in these cases of early, rapidly progressive involvement almost the only chance of recovery.

With this background, the routine treatment practiced at the Evanston Hospital can be outlined. When any respiratory difficulty continues

in a case of poliomyelitis a laryngologist is asked to see the patient. It is recognized by all the clinicians of the "polio team" that any period of anoxia is dangerous and that relief must not be postponed.

If the patient with a purely spinal type of respiratory disturbance shows respiratory embarrassment, he is placed in the respirator before any serious anoxia or exhaustion occurs. It is true that the patient coordinates better with the machine if there is considerable weakness, but even a normal person can, with careful coaching, adapt himself to the machine. The machine is set to operate at a normal respiratory rhythm, usually about 15 to 18 respirations per minute, with a negative pressure of 17 to 19 cm. of water. The patient's sense of relief may dictate slight variations of this. Positive pressure has rarely been found useful. If there is any tendency for fluid to accumulate in the throat, postural drainage with aspiration is used.

Oxygen is given from the onset, and a good fluid and electrolyte balance is maintained, parenterally if necessary. Penicillin is used routinely to combat secondary infection.

With bulbar involvement, if there is any difficulty in swallowing or breathing, the foot of the bed is elevated at least 20 degrees, the mattress is not allowed to sag, and the head is kept lower than the larynx. It is obvious from figure 2, in which the main bronchi and the trachea are outlined with a radiopaque medium, that even this angle of elevation gives hardly more than a horizontal flow of secretion from the lower part of the lung bed.

If the patient is a young child who will lie prone with the face turned to the side, drainage is satisfactory at that angle. It is a common error not to secure real postural drainage. Failure to obtain more than 5 degrees of elevation, as indicated by the illustrations found in some reports, may well explain certain cases in which the causes of anoxia were not understood. With good postural drainage my associates and I have not had serious trouble with injury of the esophageal mucosa due to gastric juice in patients otherwise properly treated.

With excess fluid gravitating to the pharynx, aspiration is usually satisfactory. This is best done, in general, by employing continuous gentle water suction, the apparatus, with a valve to prevent back flow, being attached to a rubber tube with multiple perforations, inserted through the nose to the pharynx. An alert nurse must be constantly present, and she will, in addition, occasionally have to apply mechanical suction by catheter or metal tip to secretion accumulated in the nose, the mouth or the pharynx.

Again oxygen is given from the onset, and a fluid and electrolyte balance is maintained. It is important to remember that daily from 1,000 to 1,500 cc. of fluid are lost by saliva alone,[4] so that usually for

4. Christian, H. A., in Osler, W.: The Principles and Practice of Medicine, ed. 14, New York, D. Appleton-Century Company, Inc., 1942, p. 624.

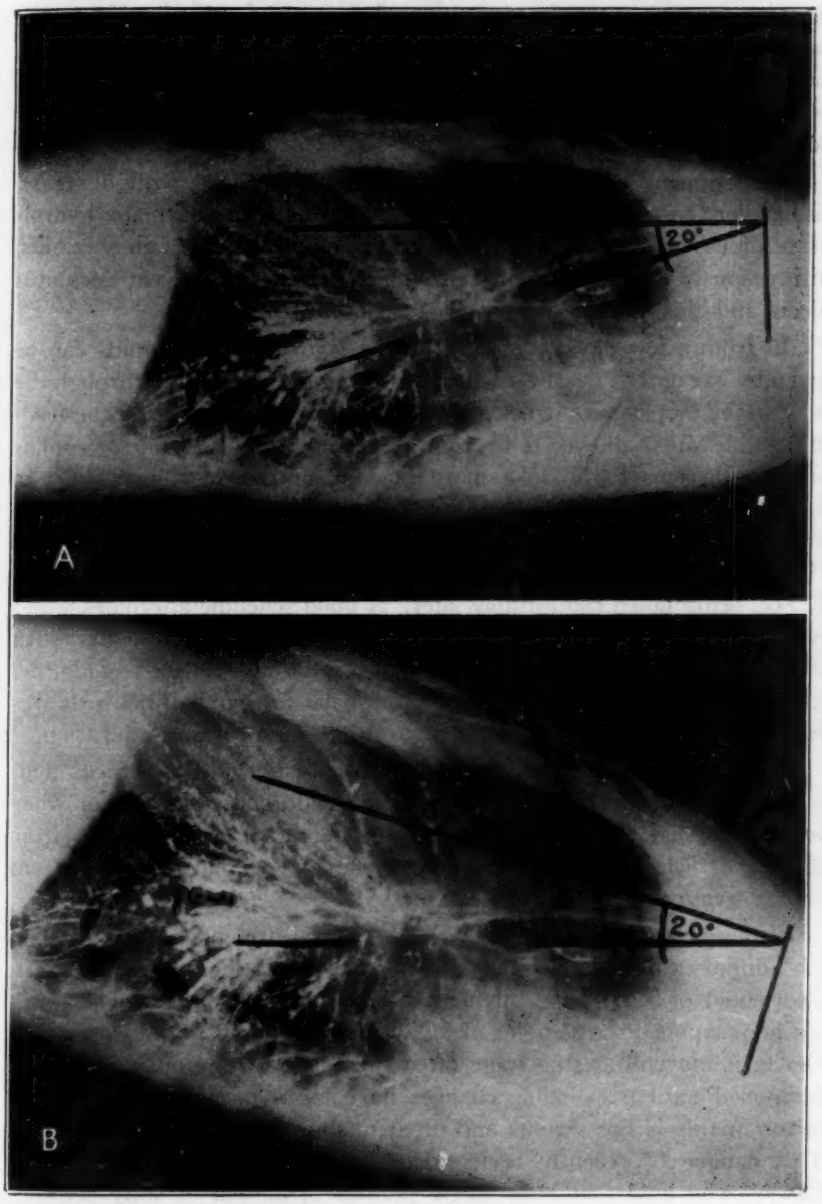

Fig. 2.—The trachea and the bronchi are outlined with radiopaque oil. *A* represents the declination of the main bronchi from the body axis. *B* shows the elevation necessary to get even slight postural drainage in this patient.

an adult from 2,500 to 3,500 cc. should be given parenterally. This fluid is necessary not only to carry on the normal functions of the body but also to maintain healthy bronchial and pharyngeal mucosa and to keep the secretion thin enough to be easily moved by action of cilia, aspiration and movement of air. For the same reason, atropine is not used.

Nourishment is maintained for the first few days with dextrose, administered parenterally. After that, "amigen" (an enzymatic hydrolysate of casein) or plasma is also used, supplemented with vitamins, intravenously injected. A good protein level helps to prevent decubitus ulcers and aids wound healing if tracheotomy is done.

In from a few days to a week, in the favorable case, liquids can be given by gavage. A nasal catheter of medium size is inserted through the nose to the esophagus. It is kept carefully closed except during the feeding, and no air is allowed to be aspirated through it. A Levine or similar long tube seems to provoke more reaction and seems more likely to induce vomiting or reflux from the stomach. Feeding is stopped at the first feeling of fulness on the part of the patient. Usually as much as 100 cc. of water is tolerated at the outset if given slowly, with 350 to 400 cc. being taken comfortably later. A good schedule alternates water every four hours with a balanced food formula every four hours. An attempt is made to give at least 1,800 calories a day. After feeding, the patient is allowed to lie with the respirator flat for one half hour, to prevent reflux, and then he is returned to the postural drainage position.

In many cases the foregoing line of treatment will not suffice. This is true especially when the patient has not had good care early or when he is exhausted, or antagonistic and uncooperative, as he may be from anoxia, or when there have been mechanical or human lapses from proper treatment.

If the patient has not improved on postural drainage, aspiration and the administration of oxygen and fluids, if he is increasingly restless, disoriented or exhausted, further intervention is necessary. This must not be postponed to the point of failing heart, marked cyanosis or coma. Tracheotomy will at this stage often prove lifesaving and must not be postponed until irreversible changes have taken place. One important factor in this is the serious and often unappreciated danger of anoxia. This danger I [5] recently reviewed, citing among others Thorner and Lewy,[6] who hold that even single periods of anoxia of sixty seconds'

5. Galloway, T. C.: The Danger of Unrecognized Anoxia in Laryngology, Ann. Otol., Rhin. & Laryng. **55**:508 (Sept.) 1946.

6. Thorner, M. W., and Lewy, F. H.: Effects of Repeated Anoxia on Brain, J. A. M. A. **19**:1595 (Nov. 6) 1940.

duration may produce distinct morphologic change of brain tissue. Windle and Becker,[7] Courville [8] and many others showed that a few minutes of anoxia may produce widespread damage of cells and necrosis. Repeated periods of hypoxia, also, as shown by Morrison,[9] may produce extensive injury of the brain. The immediately adverse effect of anoxia on the respiratory center, on vasomotor control, on the adrenal glands and on the cardiac muscles and its part in the increase of permeability of the capillaries of the lung bed must be recognized and combated early. The failure to recognize restlessness as a sign of anoxia may lead to the dangerous use of opiates or other sedatives, with further depression.

Perhaps it is even more important to realize what may follow even brief periods of anoxia even though the patient lives. Courville,[10] Barach and Rosenstine [11] and others have shown that personality changes, insanity, mental and emotional depression, and athetoid, parkinsonian and even decerebrate states may follow anoxia. Preston [12] recently reported a series of 132 cases in which anoxia occurred at or near birth: Every child suffered enough damage to affect seriously his subsequent behavior, 35 of them being left with subnormal intelligence. There should certainly be no complacency that will allow help to be withheld while the patient sinks into coma because of the fatalistic attitude that this condition is due to the virus infection. The greater effect of anoxia on brain tissue previously damaged by infection is also probably important. And it is to be suspected that some postinfectious states ascribed to virus infection or to psychologic factors are the results of periods of anoxia.

The symptoms of anoxia are not always clear in the presence of severe infection. Restlessness, disorientation and uncooperativeness are early symptoms. Instead of cyanosis in exhausted patients one sees often only gray pallor.

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7. Windle, W. F., and Becker, R. F.: Asphyxia Neonatorum, Am. J. Obst. & Gynec. **45**:183 (Feb.) 1943.

8. Courville, C. B.: Asphyxia as a Consequence of Nitrous Oxide Anesthesia, Medicine **15**:129 (May) 1936.

9. Morrison, L. R.: Histopathologic Effects of Anoxia on the Central Nervous System, Arch. Neurol. & Psychiat. **55**:1 (Jan.) 1946.

10. Courville, C. B.: Ether Anesthesia and Cerebral Anoxia: Study of Causative Factors in Serious Anesthetic and Post-Anesthetic Complications, Anesthesiology **2**:44 (Jan.) 1941.

11. Barach, A. L., and Rosenstine, E. A.: Hazard of Anoxia During Nitrous Oxide Anesthesia, Anesthesiology **6**:449 (Sept.) 1945.

12. Preston, M. I.: Late Behavioral Aspects Found in Cases of Prenatal, Natal and Postnatal Anoxia, J. Pediat. **26**:356 (April) 1945.

As Galbraith and Steinberg [13] and others have shown, and as I [14] have emphasized in discussing tracheobronchitis, atelectasis and, with secondary infection, pneumonitis and other changes speedily follow obstruction. In as little as twenty-four hours these conditions may become irreversible. This is especially important in cases of poliomyelitis, since most of those who die do so because of pulmonary complications.

Intubation may be thought of at this stage, but, aside from its obvious disadvantages, it cannot be expected to take the place of tracheotomy since it neither provides a short circuit for secretion in the airway nor easily permits deep aspiration.

What will tracheotomy accomplish? Most important is the by-pass which, by allowing air to enter into the trachea without being sucked through the pharynx, prevents secretion from being drawn into the lung field. This is easily demonstrated in the model shown in figure 1 by use of fluids of the approximate viscosity of oral secretion. The second advantage is that it permits secretion or mucous plugs to be easily removed by aspiration—supplemented by irrigation if necessary. The management after this is much simpler.

Tracheotomy has too often been looked on as a measure of last resort, and effects have been attributed to it which were due to the serious condition for which it was done or to the fact that intervention came too late. The operation is a relatively simple one, especially if done over a bronchoscope or an endotracheal tube, and, as pointed out by Jackson,[15] Negus [16] and others, the mortality of the operation is a fraction of 1 per cent. I have not seen any adverse results from it in cases of poliomyelitis except for a tendency toward drying, which can be prevented by humidification of the inspired air. The Minneapolis group performed tracheotomy on 75 patients with poliomyelitis and noted no serious results from the operation.

The patient will often breathe well after tracheotomy. He is then placed in position for postural drainage and given the usual care, including aspiration of secretion through the tracheotomy tube.

If he is exhausted, or does not breathe well, even though the disease is of the bulbar type, he is placed in a respirator. The tracheotomy should, as a rule, be fairly high—through the third or second ring—

13. Galbraith, E. G., and Steinberg, B.: The Developmental Mechanism of Pulmonary Atelectasis, Ann. Otol. Rhin. & Laryng. **46**: 800 (Sept.) 1937.

14. Galloway, T. C.: Laryngotracheobronchial Obstructions and Their Secondary Effects, Ohio State M. J. **36**:851 (Aug.) 1940.

15. Jackson, C.: Tracheotomy, S. Clin. North America **15**: 117 (Feb.) 1935.

16. Negus, V. E.: An Explanation of the Respiratory Failure Sometimes Occurring After a Successful Tracheotomy, Tr. Am. Laryng. A. **60**:92, 1938.

to allow for this possibility. The rubber collar is satisfactorily held away from the tube by a "gray iron" bar, ⅜ by ¾ inch (approximately 1 by 2 cm.), bent at a suitable angle and held under the iron flange over the neck outlet.[2] We have been routinely using this simple device with the standard respirator and rubber collar and have found it satisfactory, with only slight adroitness necessary to remove the inner tracheal cannula for cleaning.

Continuous water suction is maintained through a nasal tube with multiple perforations. Intermittent mechanical suction is done through the tracheotomy tube as needed, by means of an obliquely cut soft catheter with multiple perforations near the end. If plugs or crusts occur, irrigation with 1 to 4 droppers of 3 per cent sodium bicarbonate in isotonic solution of sodium chloride is done as previously described for tracheobronchitis. I have used hydrogen peroxide for tenacious crusts, but it seemed too irritating. If necessary for removal of secretions or plugs, the tracheotomy tube may be temporarily withdrawn and an obliquely cut soft catheter inserted through the wound down into either main bronchus, with or without previous irrigation.

Any patient with poliomyelitis in whose respiratory tract a serious accumulation of fluid occurs should, if not doing well, be treated by tracheotomy. Difficult respiration with the respirator, cyanosis, restlessness and coma are indications for further relief. Even patients with apparently purely spinal respiratory paralysis may require this, as they may have accumulations of inflammatory secretion which should be aspirated.

A patient already in a respirator may require tracheotomy. In that case a bronchoscope is passed, aspiration done, a tracheal anesthetic tube inserted, an oxygen bag attached and intermittent artificial respiration maintained. The respirator is opened out, the head is slid through the neck opening and tracheotomy is done over the rubber endotracheal tube. The procedure is inconvenient, but in such an emergency it permits the least interruption of respiration. The rubber tube is withdrawn as the tracheotomy tube is inserted, and the oxygen bag is attached quickly by a tight connection to the tracheotomy tube. A slight interruption occurs when the head is slid back through the ring, but usually little time is lost.

This procedure is difficult for the operator and adds a definite and often important element of anoxic depression or cardiac strain. Therefore, one should not wait until a desperate situation forces intervention but should anticipate it. Many patients previously were lost in respiratory obstruction because physicians were taught to wait for a rapid pulse, marked cyanosis and depression. This was the stage of imminent heart failure and irreversible exhaustion. They have greatly cut down

the mortality by earlier intervention. Similarly with obstruction due to secretion, they are coming to the same view.

Priest, Boies and Goltz [1] recently raised the question of prophylactic tracheotomy in bulbar poliomyelitis. A thoughtful house physician who was skeptical about the need and value of the first tracheotomy he had seen for bulbar poliomyelitis said, after he had followed 3 cases in which this treatment was employed, "If I get bulbar polio I want a tracheotomy right off." If the indications are not clear, one certainly must err, if at all, on the side of too early and perhaps unnecessary intervention. If this intervention proves ultimately necessary, one cannot say that it was ever too soon. The earlier case histories indicate that some of the patients who died would certainly have been saved by more timely intervention. A review of some 57 cases of bulbar poliomyelitis at the Evanston Hospital indicates that few patients who had serious breathing difficulty associated with difficult swallowing recovered without tracheotomy. It must be remembered, however, that bulbar poliomyelitis may vary from paralysis of one or two nerves through mild swallowing difficulty to the extreme complex, and tracheotomy is not advocated without definite indications. If necessary, however, it should be done while still elective and not as a forced last desperate measure.

SUMMARY

Anoxia due to accumulated secretion is a most important factor in bulbar poliomyelitis. Early and adequate care, administration of oxygen, proper postural drainage and aspiration will often suffice for recovery. With increasing signs of anoxia, further intervention must be weighed.

Although the following recommendations are perhaps to be considered only tentative on the basis of present understanding of the disease, it seems that indications for tracheotomy in poliomyelitis should be definitely set up. These are my own convictions, fortified by the thorough study of Priest, Boies and Goltz [1] of a large number of cases, and I believe them to be conservative.

The indications for tracheotomy in poliomyelitis are:

1. Fluid in the upper airway, with signs of anoxia increasing in spite of the administration of oxygen, postural drainage and aspiration. Restlessness, dyspnea, cyanosis, disorientation and mental depression are important determinants.

2. Unconsciousness or marked restlessness in a patient not previously treated and not responding to other treatment in a few minutes.

3. Marked restlessness or stupor in a patient in a respirator even if he apparently has a spinal type of poliomyelitis.

4. Fluid accumulation not otherwise easily and certainly taken care of in a patient requiring the respirator.

5. Rapidly progressive bulbar symptoms, as recommended by the Minneapolis group. Most of our patients in this group had had an onset with severe vomiting.

6. Bilateral paralysis or spasm of the vocal cords.

7. Markedly increasing signs of vasomotor failure not explained as due to a cause other than anoxia.

8. Untrained or inefficient attendants, inadequate equipment, or poor cooperation of the patient, with doubt that the airway will be kept constantly free of secretion.

Further discussion of some of these points is indicated:

If a patient under observation loses consciousness, it probably means intervention has been too long delayed.

Under the third indication, it is not always possible to diagnose the type of involvement, especially as an originally spinal involvement may quickly ascend to the bulb. Moreover, fluid may accumulate not only because of difficulty in swallowing but because of such factors as bronchial or pulmonary inflammation, increased permeability of capillaries due to anoxia or excess of carbon dioxide with increased negative intrathoracic pressure.[17]

Thorough direct examination to determine that paralysis of the abductor muscles of the larynx is the cause of obstructive symptoms is not often possible. Protective spasms to prevent self drowning may produce the same picture.

Priest and his colleagues emphasized the importance of circulatory collapse, indicated by dusky red appearance, rapid irregular pulse and low blood pressure, as an indication for intervention.

Uncooperativeness, while it may be a matter of temperament, is in my experience much more often a sign of important anoxia, which makes it all the more important to help the patient in spite of himself.

Tracheotomy is more necessary when the attending team has had little experience with the disease. If all the personnel were thoroughly trained and the mechanical arrangements perfect, tracheotomy might be seldom required. But unforeseen failures occur too easily. For instance, an assistant substituted, against orders, a fine medicine dropper tube for a proper glass connecting tube and thus prevented proper aspiration, which made an emergency tracheotomy necessary. In another instance an untrained attendant relieved the experienced nurse. Poor aspiration during two half-hour periods led to anoxia from which a decerebrate state developed.

This discussion is given from the point of view of the laryngologist and is set forth rather categorically in order to focus attention on the

17. Drinker, C. K.: Pulmonary Edema and Inflammation, Cambridge, Mass., Harvard University Press, 1945.

factors stated. In the Evanston Hospital group, directed by Dr. Martin Seiffert, there was complete cooperation between neurologist, pediatricians, orthopedic surgeons and anesthesiologists. The laryngologist plays, perhaps, a minor part in the "polio team" but he must be prepared to bear his share of responsibility. It is only fair, also, that he be allowed to see the patients early and that his special experience be used to assess properly the obstructive and anoxic factors, to note progress and to intervene if necessary before serious damage has been done.

The treatment outlined may be applied to other conditions. It was recently used successfully in a presumed case of myasthenia under neostigmine therapy in which toxic spasm, accumulation of fluid and coma developed presumably from anoxia.

636 Church Street.

CHRONIC PURULENT OTITIS MEDIA

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I MAKE no apology for offering a paper under this title. More papers have been written on this subject than on any other in otology. The importance of the subject is paramount. From the time of Hippocrates chronic purulent otitis media has been a disabling, crippling, disgusting and death-dealing disease. It has been the despair of otologists throughout the ages. This is not to say that otologists have not obtained cures in a large percentage of patients, but it is to say that in spite of the most meticulous care there has always been a percentage, at least 20, that have been given up as incurable and so advised by conscientious otologists.

INCIDENCE

The number of cases of chronic purulent otitis media in the world today is truly appalling. Arthur Cheatle examined 1,000 school children in England and found 88 cases, 8.8 per cent.[1] Among the 2,074 school children of Essex the percentage was found to be 4.8. The report of the Surgeon General of the United States to the Secretary of War in 1919 gave the incidence of chronic otorrhea among officers as 5.2 per thousand. Among the enlisted men the incidence was 12.23 per thousand.

The first point I wish to make in this paper is the widespread incidence of chronic purulent otitis media. It is found in about 2 per cent of the population. There are 140,000,000 people in the United States. One can estimate that there are 2,800,000 cases of this disease in the United States. Every one of these cases is the problem of the otologists. The next thing to consider is what are the otologists doing to solve this problem? In other words, are they waging a successful warfare against this disease which is crippling and killing so many people the world over? I think all of them will agree that the most optimistic among them has the opinion that so far their record of cures is not impressive. People are dying with abscess of the brain, meningitis and thrombosis

Read at the meeting of the Southern Section of the American Laryngological, Rhinological and Otological Society, Inc., at Miami Beach, Fla., Jan. 6, 1947.

1. Lishman, F. J. G.: Brit. M. J. **2**:1165 (Dec. 11) 1937.

of the lateral sinus the world over. If the deaths are numerous in proportion to the number of cases involved, what about the 2,800,000 who have to live with this loathsome disease? They are humiliated and depressed by the foul odor of the discharge. They are deafened by the distressing ravages of the disease. They are rendered unfit for society and for occupational employment and are rejected by insurance companies and so are deprived of the protection of insurance against disease and disability. The insurance companies with their scientific actuaries found out before the otologists did the crippling, death-dealing menace of the discharging ear. Is it not time that otologists recognize the seriousness of this problem and appreciate that it is their sole responsibility? I should like to quote some world authorities on the gravity of the situation and the failure of otologists to make a dent in it so far. Every otologist has his technic in dealing with the chronically discharging ear. The fact that there are almost as many methods as there are otologists bears eloquent testimony to the fact that few of the methods are satisfactory.

In 1933 Downey,[2] professor of otology in the University of Maryland Medical School, published a comprehensive paper on chronic suppurative otitis media. He wrote:

> . . . Now and again it is well to take an inventory of our own and the experiences of others, regarding those diseases which though not uncommon are always baffling and difficult to treat successfully. Excluding oto-sclerosis and its kindred condition, the chronic suppurative ear presents the most exasperating problems of clinical otology. Its very chronicity may be the evidence of failure after the most careful preventive measures; it is always, if I may speak in metaphor, a challenge to both medical and surgical combat before which one is just as likely as not to go down in defeat.

To a person so afflicted such an ear can or may be (1) a menace to life by extension of the inflammatory process to the labyrinth, the meninges, the lateral sinus, the cerebrum or the cerebellum, (2) a frequent cause of permanent reduction of hearing, (3) a constant source of annoyance and esthetic embarrassment and (4) an economic handicap not only from the standpoint of occupation but from that of insurance, for it may prevent the person from obtaining protection or may increase the premium to a prohibitive degree.

Banham,[3] squadron leader in the Royal Air Force, reported that 20 per cent of some 3,500 new patients attending a Royal Air Force Clinic were found to have active chronic otitis media. An equal number were found to have symptoms resulting from past infection of the middle ear. Some 40 per cent of all attending the clinic had, therefore, symptoms of active or inactive middle ear infections. Apparently a similar

2. Downey, J. W., Jr.: West Virginia M. J. **30**:97 (March) 1934.
3. Banham, T. M.: J. Laryng. & Otol. **59**:117 (April) 1944.

percentage is found in the other services. Craig [4] reported that 25 per cent of his patients at a military hospital were found to have chronic suppuration of the middle ear. It will be seen, therefore, that the treatment of disease of the middle ear occupies a large part of the service otologist's time.

Hitschler and Vastine [5] said:

> We conclude that the roentgenographic studies of this group have not added materially to our information except in a negative way; i. e., they eliminated abscess formation. Ashley said that roentgenograms are of little value in diagnosis because sclerosis is so frequently present.
>
> Nasal examination of the 16 patients showed the nasal structure to be normal in 12. There was septal deviation in 4. There was no demonstrable relationship between septal deviation and persistence of aural discharge. The nasal mucosa appeared normal in 5 children. It was reddened to a greater or lesser extent in 11. Nine children had pus in the nose and 2 an excess of mucus. Transillumination indicated clouding of one or more sinuses in 4 patients. In each of the 4 one antrum was denser than the other, while involvement of 2 frontal sinuses was indicated by this examination. The findings by transillumination and those by roentgenography often did not agree. One maxillary or one frontal sinus often showed more clouding by transillumination but less involvement by roentgenography, or vice versa. The roentgenologist reported clouding in 3 frontal sinuses and 4 sphenoid sinuses, with possible exudate in 1 of the sphenoid sinuses. There was clouding of the ethmoid in every child, although it was slight in 6 cells. One or both maxillary sinuses appeared pathologic in 13 of the children. In 6 of these the antral findings were such as to indicate the probable presence of exudate. In 5 of these 6 the antrums were irrigated, and exudate was found in 3. It must be noted that there was at least a six week period between the taking of films of the sinus and the antral irrigations.

With regard to the report of the 16 cases of Hitschler and Vastine, it is pretty clear that practically every one of the 16 patients had sinus involvement of one kind or another. It is my firm belief that if these children had been given the benefit of surgical drainage their purulent otitis media might have been cleared up and their hearing and health restored.

In the Royal Air Force examination of 35,000 aviators, it was disclosed that there were 1,750 men in the service who were absolutely useless to the army and the navy. The ranking officers of the armies and navies of Great Britain and the United States stated positively that these men were useless to the armed services.

Brown Kelly,[6] reporting before the Royal Society of Medicine, May 4, 1945, said that the point which struck him was the enormous incidence of aural disease in the naval hospital, which accounted for 30 per cent of the cases dealt with in the hospital. The disease was serious

4. Craig, D. H.: J. Roy. Army M. Corps **76**:86 (Feb.) 1941.

5. Hitschler, W. J., and Vastine, M. F.: Chronic Suppurative Otitis at Pennsylvania School for Deaf, Arch. Otolaryng. **43**:226 (March) 1946.

6. Brown Kelly, H. D.: J. Laryng. & Otol. **59**:333 (Aug.) 1945.

in the navy, for a sailor with chronic purulent otitis media had to be treated as an inpatient.

Formby [7] said before the Royal Society of Medicine, May 4, 1945, that the cases studied in the ear departments of military hospitals during the past three years showed on analysis that almost exactly a quarter of the work had been in dealing with chronic suppurative otitis media. Taking the persons reporting to medical officers and camp reception stations and the so-called first aid posts for the army all over the country during 1943, there were approximately 1,000 reporting every week on account of their ears, just over 50,000 a year, and of these more than half had chronic suppurative otitis media.

An interesting report as regards the incidence of chronic purulent otitis media is that of Guthrie,[8] aural specialist to the Scottish command. It is interesting from the fact that the examination was made during peacetimes from the year 1927 through 1935, a period of nine years. During this period, 2,931 new patients were examined at the military hospital at Edinborough Castle. An examination of the record reveals the fact that two diseases, or rather groups of diseases, far outnumber all others in frequency. They are (1) tonsillitis and (2) suppuration of the middle ear (otitis media suppurativa) as set forth in the accompanying tables: The tables show that of the 2,931 recruits examined, better than 28 per cent had chronic purulent otitis media. He stated further that these tables show that diseases of the middle ear (usually chronic suppurative otitis media) are the principal cause of unfitness in recruits and account for the rejection of from 50 to 60 recruits in every thousand who apply.

Miller [9] has given an interesting report of observations which he made while a member of the otolaryngologic staff at Fort Bragg, N. C. There were 125,000 troops stationed there. It is interesting from the fact that these troops were supposed to be already screened out. It is further interesting from the fact that these were healthy men between the ages of 21 and 27. At that time the regulations were rather strict as regards otitis media. Yet at that post actually 4 per cent of all rejections were due to otitis media in some form, nearly all being due to chronic purulent otitis media.

TREATMENT

Persky [10] said:

Acute or chronic sinusitis also plays a role in the reactivation or maintenance of a chronic suppurative otitis media. Meticulous attention to these sinuses is

7. Formby, M.: J. Laryng. & Otol. **59**:334 (Aug.) 1945.

8. Guthrie, D.: J. Roy. Army M. Corps **67**:36 (July) 1936.

9. Miller, J. B.: Paper read before the Nashville Academy of Ophthalmology and Otolaryngology, Jan. 20, 1947.

10. Persky, A. H.: Eye, Ear, Nose & Throat Monthly **24**:227 (May) 1945.

of prime importance. If conservative treatment is not sufficient, surgery should be employed. It stands to reason that any of these causes which will help reactivate the otitic infection through the eustachian tube, will influence any form of otitic therapy.

Banham [8] reported the result of conservative treatment of 200 patients; 167 had dry ears after one week to three months of treatment, leaving 33 with running ears, a percentage of 16.5. He made a significant statement, however. Of all the 200 treated, in only 14 did the perforation close. My experience would lead me to believe that at any time these patients with unhealed perforations caught cold, purulent discharge was liable to set in.

Rottboll Hansen,[11] of Copenhagen, Denmark, contributed an interesting article to the *Transactions of the Ninth Nordic Congress of Oto-Laryngology*. It is a follow-up investigation of 147 children who because of otitis had been admitted to the ear department of the Kommune Hospital in 1928. The study was made ten years later. Nine of the children had died, 4 as a result of acute aural disease, while in the hospital. The after-examination was made on 138. Of this number, 50 showed the ear healed without operation other than paracentesis; 68 were operated on; 20 were left with chronic suppuration. Thus, after ten years 16 per cent were left with chronic purulent otitis media.

Thirty-nine had been treated only with paracentesis. Eighty per cent of these recovered. However, 25 per cent had recurrent discharge from the ears later; 1 child had to be operated on five years later.

Group 2 consists of 11 children; 66 per cent of these had recurrent discharge, some right up to the examination ten years later.

Group 3 consists of 30 children who underwent mastoidectomy; 10 had to be operated on again or several times; 10 had suppuration of the middle ear once or several times after the operation.

Group 4 consisted of 28 children who were operated on but did not recover normal hearing. Seventy per cent of this group had repeated suppuration of the middle ear, some right up to the time of examination. Ten of these were operated on as many as four times; several of them were operated on the year of the examination, or nine years after hospitalization.

Group 5 comprises the group of 20 who were found to have chronic suppuration of the middle ear after ten years. Two of these were submitted to the radical mastoidectomy and were dismissed from the hospital as having healed ears. Six others were dismissed as healed, though later chronic purulent otitis media developed. Two other patients in this group underwent simple mastoidectomy. Both these

11. Rottboll Hansen, B.: Acta oto-laryng., 1939, supp., p. 201.

patients reported frequent discharge. The other 4 patients had almost continuous treatment with irrigations and the like, and examination revealed fetid pus and granulations. The 12 remaining patients had chronic otorrhea when they entered the hospital. All 12 of them have undergone radical mastoidectomy. After ten years there were 7 with constant fetid discharge. The hearing of all these was much impaired. The whole picture presented by more than half the group was a dark one and illustrates graphically the problem of dealing with acute and chronic purulent otitis media. Rottboll Hansen makes the pertinent observation that otologists must be skeptical with regard to otitis in children who have had otorrhea for a long period even if the ears become dry after a brief stay in the hospital.

So far as I know this is the only investigation carried out on a group of patients who had been hospitalized for otitis media in childhood ten years afterward and to my mind is a most valuable and instructive study. However, I feel strongly that if these children had been examined with respect to their sinuses and proper drainage instituted, in all probability all might have been cured.

I examined with great care the fourth edition of Turner's [12] article on chronic purulent otitis media. I did so because I have great respect for Logan Turner. He published the article about the time that I published my first article on the association of sinus disease and infections of the middle ear and mastoid process. He reported 42 cases of mastoiditis in 28 of which there was chronic sinus infection, mostly on the same side as the mastoiditis. I was greatly disappointed when he said nothing whatever about the association of sinus disease and chronic purulent otitis media. He devoted his entire article on treatment to local measures to be carried out in the ear and operations on the mastoid process. Evidently he never followed up his first observations.

In the otologic section of the Royal Society of Medicine, May 7, 1937, there was a long discussion of otitis media occurring in early childhood (under 5 years). Le Mée,[13] of Paris, France, made this pertinent statement in discussing postmortem examinations made on infants dying with otitis media:

> . . . secretions will also be found in the two sinuses existing in the infant, i. e., the maxillary sinus and the ethmoid sinus. Why then is otitis always considered to be the cause of death, not sinusitis, which is just as serious a disease? It is because, when doing an autopsy, one generally does not examine the sinuses. This precaution is also forgotten on examination of the living patient.

12. Turner, A. L.: Chronic Purulent Otitis Media, in Diseases of the Nose, Throat and Ear for Practitioners and Students, ed. 4, Baltimore, William Wood & Company, 1936.

13. Le Mée, J. M., and others: J. Laryng. & Otol. **52**:685 (Oct.) 1937.

Ebbs,[14a] in the same discussion, reported on the analysis of postmortem observations of the middle ear and the mastoid process made on 880 children varying in age from birth to 14 years. These observations were made in consecutive examinations of almost 100 per cent of the children who had died in a children's hospital from all causes during four years—a hospital which receives patients from a large area in the midlands. I take it that the hospital is in Birmingham, England. They were all examined to determine the presence or the absence of infection of the middle ear and the mastoid process. Purulent material was found in one or both ears in 528. This is startling. It shows that more than half of the children who died in this hospital from all causes were found to have a purulent infection of the middle ear and the mastoid process. Of 603 children under 1 year of age, 368 had purulent otitis media when they died, or 61 per cent; of children in the second year of life, 52 per cent, and of those of other age groups, 30 per cent. An investigation of the sinuses was made in the 1 year group. Of 496 children in the 1 year group, 280 were found to have purulent otitis; 42.5 per cent of the 280 also had sinusitis.

Ebbs quoted an article by Crooks, of the Hospital for Sick Children, Great Ormond Street, London, stating that in the examination of 100 children 24 per cent were found to have infected antrums. These were children of an older age group who were having tonsils and adenoids removed. Ebbs said that this important study illustrated the frequency of the disease. Crooks also reported that 44 of 100 children examined for sinusitis had purulent otitis media.

Ebbs makes the interesting observation that he almost invariably finds purulent otitis, mastoiditis and sinusitis in postmortem examinations of children with enteritis. Most interesting is his finding in postmortem examinations of children dying with pneumonia.[14b] He divides these into two groups, those with primary and those with secondary pneumonia. The first group come into the hospital with a confirmed diagnosis of pneumonia. The second group come in with some other important condition but have pneumonia as an associated condition or have been found to have pneumonia at autopsy; 71.6 per cent of the first group (134) had associated otitis media at autopsy, while 70 per cent of the second group (229) also had otitis media. Ebbs asked "What is the significance of this?" and quoted Campbell (1934), who studied 130 young children with pneumonia, 70 per cent of whom had otitis media and 100 per cent had sinusitis. He quoted Cullom (1934), who expressed the opinion that infection of the upper respiratory tract is a "menace to all lower lying structures." "Thus," Ebb said, "it may lead

14. Ebbs, J. H.: (*a*) J. Laryng. & Otol. **52**:691 (Oct.) 1937; (*b*) Proc. Roy. Soc. Med. **30**:1407 (Sept.) 1937.

to tonsillitis, bronchitis, bronchiectasis, pneumonia, and severe gastro-intestinal disturbances." Ebbs then commented:

> . . . Whether otitis media or mastoiditis is a significant factor in the aetiology of pneumonia or not, it is apparent to me that the high percentage of children who die with pneumonia and have an infection of the ear, is very strong evidence in favour of a preceding, concurrent, or secondary upper respiratory infection. The fact that nearly all of these children were found to have broncho-pneumonia rather than lobar pneumonia is further evidence of it being secondary to, or associated with, upper respiratory infection.

At the forty-eighth annual meeting of the American Laryngological, Rhinological and Otological Society Inc., in 1942, there was presented a comprehensive article on "Chronic Otitis Media" by Moorhead.[15] In it there is not one reference to the association of chronic suppuration of the middle ear with sinus disease.

Bond Hill seems to have considered that it is only nasal obstruction and adenoids that are causative factors in "chronic purulent otitis media." He referred to "nasal-pharyngeal disease," by which he evidently meant adenoid vegetation.

Lederer,[17] in a long discussion of treatment, divided patients with chronic purulent otitis media into four groups. For group 3 he advocated removal of osteomyelitic portions of the ossicles (ossiculectomy). For group 4 he advocated surgical intervention, simple mastoidectomy and radical mastoidectomy, and then, under the heading "General," first the removal of foci of infection, such as the tonsils and adenoids, and treatment of the paranasal sinus disease. He did not indicate that he means operation on the nasal sinuses—only treatment.

Peters[18] said: "There are few diseases today that are causing more chronic loss in the way of physical defects than acute and chronic suppurative otitis media with its many complications and sequelae." Treatment should be directed toward the throat and the sinuses—in the way of eliminating the primary foci, if necesary, in perfecting a cure.

Crooks,[19] of the Hospital for Sick Children, Great Ormond Street, London, said that the most frequent cause of otitis media is adenoids. He then said:

> The next most frequent cause . . . is infection of the accessory nasal sinuses. . . . If an infected antrum is discovered it must be washed out. . . . The cessation of ear discharge following the removal of a focus of infection in the nose or throat is often dramatic.

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15. Moorhead, R. L.: Tr. Am. Laryng., Rhin. & Otol. Soc. **48**:176, 1942.

16. Footnote deleted by the author.

17. Lederer, F. L.: Diseases of the Ear, Nose and Throat: Principles and Practice of Otorhinolaryngology, ed. 2, Philadelphia, F. A. Davis Company, 1939.

18. Peters, A. L.: New Orleans M. & S. J. **87**:90 (Aug.) 1934

19. Crooks, J.: Practitioner **141**:594 (Nov.) 1938.

Daly [20] said:

. . . In discussing a paper by Cullom in Miami before the section of the S. M. A., 1929, I reported two cases of mastoiditis cured by operation on the sinuses. My own cases and Dr. Cullom's paper made a deep impression on me. Since then I have made it a rule to examine the sinuses of every student who comes to me with acute or chronic otitis media. Frequently the involvement is on the same side.

Johnston [21] said:

. . . Infection of the sinuses is a common cause of chronicity of otitis media and I do not consider that one can hope to obtain a permanent cure of the ear unless the sinuses, particularly the maxillary sinus, have been cleared of infection. It is my impression that over half the patients with an anterior perforation have pus in the maxillary sinus often on the same side. In this connection I would like to stress the fact that anterior and posterior rhinoscopy and transillumination will often fail to reveal any abnormality. A good radiograph may be the only positive finding apart from proof puncture returning pus. I consider that in such cases, if the antral washout is clean, an opacity in the radiograph indicating thickening of the mucosa due to allergy or subsiding infection, can be disregarded. Chronic otitis media due to a solitary infection of the ethmoid is less common and of the sphenoids rare. I have never seen an otitis media with an infection confined to the frontal sinus.

I am in thorough accord with this sound and forthright statement.

Hill [22] published an exhaustive article entitled "The Chronic Discharging Ear," in which he stated:

As the middle ear infection may be kept active or periodically be reactivated by some focus in the nose, sinuses, mouth or throat, the elimination of such foci, if possible, should be an important step in treatment.

I examined more than two hundred and twenty-five papers in preparation for this paper. In practically all of them the authors devoted their entire attention to local treatment of the ear. When local treatment fails, as it often does, they then advise surgical intervention. To them surgical treatment means mastoidectomy, either the simple or the radical operation. When mastoidectomy fails to cure the purulent discharge, they classify the case as "a failure" and take comfort in the fact they have protected the patient from intracranial involvement. Those who rely on local treatment depend on cleansing by either the wet or the dry method, insufflation of powders, use of caustics, snaring out polypi, ossiculectomy and finally the simple mastoidectomy, the modified radical mastoidectomy of Heath or Bondy or the classic radical mastoidectomy.

Those who use the local treatment report a large percentage of cures. I presume all otologists recognize how deceptive percentages of

20. Daly, L.: South. M. J. **29**:1001 (Oct.) 1936.
21. Johnston, C. M.: J. Laryng. & Otol. **60**:303 (Aug.) 1945.
22. Hill, F. T.: Dis. Eye, Ear, Nose & Throat **1**:112 (April) 1941.

this kind are. I presume every one may recall plenty of cases in which he has been able to stop the drainage and then the first time the patient catches cold the discharge of the ear or ears returns. The continental otologists have followed up such cases and have found a great many of what they call recedivation cases or cases of recurrent discharges. The patients are the despair of the otologist. They return to plague him. But no doubt, a great many of them do not return. They are the victims of despair themselves and resign themselves to their running ears.

I think I have found the cause of this type of running ear, and I think I have found the remedy. In this city, in 1929, I [23] presented my first paper addressed to members of the medical profession in general on the "Etiology of Middle Ear Suppuration," in which I advanced the thesis that abscess of the middle ear and mastoid process is the result of purulent sinusitis. I showed roentgenographic slides depicting an abscess of the mastoid process and slides showing a purulent sinus on the same side. This paper was received in an unexpectedly sympathetic manner, and a number of men recalled cases that bore out my findings. Since then I have read a number of papers, four before the section of otolaryngology of the American Medical Association and four before the section of the Southern Medical Association, continuing and expanding the subject until now I have come before you to report the most amazing and exciting discovery of all. If what I have found out is true, it means a chance for health and happiness to millions of people. It likewise means work enough to absorb a good part of the time of all the otologists in the world. I have got far enough to feel that in the great majority of cases chronic purulent otitis media is dependent on chronic purulent sinusitis usually on the same side as the purulent otitis media and that radical surgical drainage of the chronic purulent sinusitis will clear up the chronic purulent otitis media in a dramatic way and in a short time. The clearing up of the chronic purulent sinusitis is followed by the formation of a new drum membrane and the restoration of hearing. And what is equally gratifying is the restoration of the patient to health, vigor and happiness.

I think that practically all otologists are in agreement now that purulent otitis media and mastoiditis are the result of purulent sinusitis. In his most comprehensive study of the sinuses Van Alyea [24] said that rhinologists commonly associate diseases of the middle ears with sinus infections. A wide range of conditions from simple tubal catarrh to acute mastoiditis may be traced to or associated with sinus disease. In support of this, he quoted two papers of mine read before the

23. Cullom, M. M.: South. M. J. **23**:1091 (Dec.) 1930.

24. Van Alyea, O. E.: Nasal Sinuses: An Anatomic and Clinical Consideration, Baltimore, William Wood & Company, 1942, p. 200.

Section of Laryngology, Otology and Rhinology of the American Medical Association. He also quoted my belief that sinus infections associated with contagious diseases become chronic and cause subsequent acute attacks.

Now what would be the logical approach to this problem? Suppose one is confronted with a case of chronic purulent otitis media. Should one not say, "This discharging ear was originally caused by a purulent sinusitis?" Should one not then ask whether, that being so, one should not investigate the sinus to see if it is still infected? I think that is a logical conclusion. One takes a roentgenogram of the sinuses and finds one or both sinuses infected. Should one not then say that these sinuses were the original cause of the infection and that they are still actively infected? Is not the next logical step the thorough surgical drainage of the sinuses?

I want to say that nothing in my surgical experience has been more dramatic than the cessation of the discharge of pus from the ears, the reformation of the new drum membrane, the restoration of hearing and the return of the patient to health and vigor. Some of these patients had had running ears from childhood, most of them for many years. Nothing in my life has filled me with such wonder as the ability of nature to repair damage, to restore function and health when the burden was lifted from her shoulders.

It has been said that it seems incredible that an ear that has been discharging pus with a foul odor for years could clear up in a short time. I was asked if this was what I meant? This is exactly what I do mean. Odor is the hallmark of a discharge of the ear associated with chronic purulent sinusitis. To my mind the odor in such an ear is not from the pathologic process in the ear itself but is due to the foul pus that is coming from the chronic purulent sinusitis. I have come to regard the eustachian tube and the middle ear in such cases as a sewer draining off the foul pus from the sinus through the eustachian tube and the middle ear. When the drainage from the sinus is stopped by surgical drainage, the drainage from the ear stops automatically. The mucous membrane of the sinuses and the ears will take a lot of punishment as long as the drainage is unobstructed. If the drainage is obstructed sufficiently long, destruction of bone sets in as a result of pressure necrosis. I found that out in my work on frontal sinuses. In 4 cases of complete obstruction of drainage there was a large area of the posterior wall that had undergone necrosis, exposing a large area of the dura. This area was ignored, and no symptoms ever resulted. The other two cases resulted in fistulas from one frontal sinus to the other, there being no drainage from that frontal sinus into the nose.

I do not claim that every ear whose discharge appears to be associated with chronic purulent sinusitis will clear up after surgical drain-

age of the sinus, but I [25] do say that all I have encountered so far have cleared up. In order to get the information I always have roentgenograms made of the mastoid processes. In a few cases involvement of the mastoid process has been shown. In all of these the involvement has cleared up after a short time. The improved drainage into the middle ear accounts for this, I think.

The aural discharge that is shown not to be associated with chronic purulent sinusitis and involvement of the mastoid process is the type that should clear up under local treatment.

As I stand before you today I feel that I am pleading for those millions on millions who are the victims of disgusting disease which sets them off from society and cripples them industrially.

REPORT OF CASES

CASE 1.—Mrs. G. W. S., aged 26, consulted me for chronic purulent otitis media of the right ear on Feb. 21, 1944. Her right ear had been discharging since early childhood. She had had constant treatment since the beginning. The right canal was filled with pus with a most foul odor. There was a large perforation in the anterior portion of the drum. The patient was depressed over the condition. She did not hear a watch on contact. A roentgenogram of the sinuses showed the right antrum dark. I did a Caldwell-Luc operation on the right antrum at St. Thomas Hospital on February 23. I saw the patient the next morning. The first thing she said was, "Doctor, there has been no discharge in my ear since the operation." Inspection showed that this was true. There was no discharge from the ear thereafter. On November 6 it was noted that there had been no discharge since the operation and that the drum membrane had reformed. An audiogram showed the hearing as good in the right ear as in the left. She has been seen at intervals since, and there has been no discharge, and hearing in the ear is practically normal.

CASE 2.—On May 3, 1945, A. K., a white man aged 25, consulted me for a chronic discharge of the right ear. Four years ago prior to this he underwent mastoidectomy. For the last two years the right ear had discharged pretty constantly. There was a large perforation in the anterior portion of the right drum membrane. He heard a watch at 2 inches (5 cm.). There was an unusually foul odor from the discharge. A roentgenogram showed the left antrum cloudy but the right antrum clear. I did a Caldwell-Luc operation on the left maxillary sinus. The drainage from the right ear ceased in a few days and has never recurred. The drum membrane reformed, and the hearing returned to practically normal.

CASE 3.—On July 5, 1946 Mrs. P. S., aged 28, consulted me for a chronic purulent discharge of the left ear. She had had intermittent discharge from the ear since childhood. The discharge had an offensive odor. She could not hear a watch on contact in the left ear. A roentgenogram made by Dr. Shoulders showed a cloudy left antrum. The roentgenogram also disclosed some cloudiness of the mastoid cells. A few days later I did a Caldwell-Luc operation on the left antrum. The antrum was filled with foul pus and polypi. The drain-

25. Cullom, M. M.: South. M. J. **30**:785 (Aug.) 1937.

age from the left ear was markedly reduced at once but did not cease entirely for several weeks, which I attributed to the involvement of the mastoid process and the foul drainage from the antrum. However, in a month's time the discharge from the ear had ceased. I saw her recently. There had been no recurrence of the discharge. The large perforation of the anterior portion of the drum had closed. The hearing of the ear was practically normal, and there was a truly remarkable improvement in her health.

CASE 4.—On Oct. 3, 1946 I was consulted by J. S., aged 25. He had undergone an illness the previous January, which left him with a chronic discharge

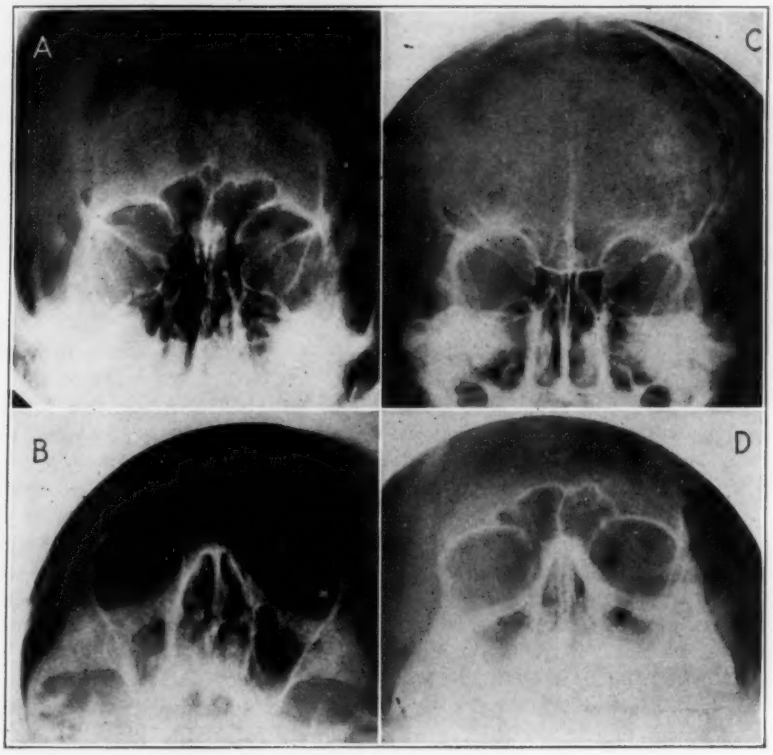

Fig. 1.—Roentgenograms taken in cases 1 to 4. *A* (case 1) shows a cloudy antrum on the same side as the chronic purulent otitis media—the right side.

B (case 2) shows a cloudy left antrum though chronic purulent otitis media was on the right side. (This film was reversed.)

C (case 3) shows a cloudy antrum on the same side as the chronic purulent otitis media—the left side.

D (case 4) shows both antrums cloudy. Chronic purulent otitis media was present in both ears.

of both ears. He had remained in the hospital for some time and had constant treatment but to no avail. At the time I saw him he had purulent drainage of both ears and also external otitis of both ears. He heard a watch at 14 inches (10 cm.) in the right ear. He could not hear a loudly ticking watch on

contact in the left ear. There were anterior perforations in both drums, the larger one in the left. Roentgenograms showed both antrums cloudy, the left more than the right. I advised radical operation on both antrums. After some hesitation and the summoning of his father from New York, the surgical treatment was agreed on and a bilateral Caldwell-Luc operation was done at St. Thomas Hospital on October 10. The left antrum was found filled with pus and polypi. Polypi were also found in the right antrum but not to the same extent. There was some drainage of the ears during his stay in the hospital. He had some reaction and was given penicillin for five days, until his temperature was normal. When he returned to the office, eleven days after the operation, the discharge of his worse ear, the left, had entirely ceased. The discharge of the right ear ceased in about a week. The perforations of both drum membranes closed rapidly after the discharge stopped. In two weeks there was complete closure of the large perforation of the left membrane, and this ear presented the appearance of a normal ear. He could then hear a watch at 30 inches (76 cm.). The hearing returned more slowly in the right ear. A recent audiogram showed the hearing normal in both ears. After three months there has been no recurrence of discharge. The perforations were closed, the drum membranes appeared normal, the hearing was normal and the health was completely restored.

CASE 5.—R. W., aged 18, consulted me on Nov. 2, 1946. He had bilateral purulent otitis media, from which he had been suffering for months. Lately he had been suffering acute pain which was, no doubt, due to external otitis in both ears. The discharge from his left ear was dark and foul in odor. He had the appearance of being ill and had lost 20 pounds (9 Kg.) in two months. I tried local treatment, which relieved his pain. As soon as the pain was relieved, he stopped coming. After several weeks he returned with a complete relapse. I had previously advised that a roentgenogram of his sinuses be made, which he declined. Transillumination indicated dense clouding of the left antrum and to a less degree of the right. A roentgenogram made by Dr. H. S. Shoulders indicated that the right antrum contained polypi, but there was uncertainty about the left. I advised that a Caldwell-Luc operation be done, which was performed at St. Thomas Hospital, with the patient under general anesthesia. I had the feeling that the left antrum was the worse and opened that first. As soon as I made an opening in the anterior wall, a mass of polypi showed up. I removed such a mass of polypi that it seemed remarkable that the antrum could hold them. The right antrum contained a large amount of polypoid growth also. On account of the mass of polypi and the infection, I prescribed penicillin, 40,000 units every four hours. In spite of that he had a septic temperature for a week; then his temperature came down to normal. The discharge of the right ear ceased at once and I thought the left ear had ceased to run, but a slight discharge showed up after a few days, though nothing to compare with the profuse foul discharge previous to the operation. The roentgenograms of his mastoid processes indicated involvement of the cells of the left mastoid process, which indicated to me that broken-down cells of the mastoid process were contributing the small amount of discharge that was still seen.

As soon as his temperature became normal, the improvement in his general condition was remarkable. His color changed, his appetite returned and he

began to take on weight. It will, of course, be an uphill climb to get back his normal health and vigor, but he is going uphill now instead of down.

One of the remarkable features of this case is that the sinuses could have got into such a wretched condition without ever attracting his or any one else's attention. When he began to run down, he went to an internist for a general check-up. No examination was made of his sinuses, and the internist's report was essentially negative. However, that is characteristic of this type of case. Not a single one of the 17 patients under consideration had ever had the sinuses examined. This demonstrates conclusively that destructive sinus disease can exist without attracting the attention of the patient or the physician. The patient was seen Jan. 18, 1947. There was still some drainage from the left ear. The patient had gained 18 pounds (8 Kg.) in four weeks. He had a ravenous appetite.

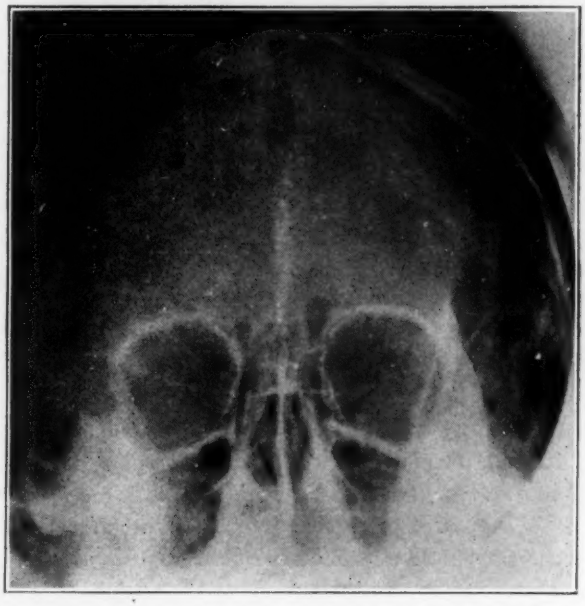

Fig. 2 (case 5).—The roentgenogram shows both antrums filled with polypi. Chronic purulent otitis media was present in both ears.

SUMMARY AND CONCLUSIONS

Statistics are quoted which show that chronic purulent otitis media has a worldwide incidence of great proportions.

The disease inflicts a great loss in health and economic efficiency and has a serious mortality rate.

Physicians are agreed that the cause lies in purulent sinusitis.

Physicians are likewise agreed that the great number of cases of purulent sinusitis is the aftermath of the acute exanthematous diseases of childhood and epidemic influenza.

The thesis of this paper is founded on the assumption that chronic purulent sinusitis is the activating cause of chronic purulent otitis media.

The conclusions of this paper are: (1) that in the great majority of the cases the chronic purulent otitis media can be cleared up, the perforation of the drum can be closed, the function of hearing and the health of the patient can be restored by surgical drainage of the purulent sinusitis; (2) that in those cases in which the mastoid process has become involved the drainage of the purulent sinusitis will greatly facilitate the measures taken to clear up the involvement of the mastoid process; (3) that as long as the activating purulent sinusitis is left undrained, local treatment will prove unavailing and time consuming.

The manner in which chronic purulent otitis will clear up following surgical drainage in a suitable case is startling and dramatic.

700 Church Street.

A BRIEF HISTORY OF OTOLARYNGOLOGY IN THE UNITED STATES FROM 1847 TO 1947

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SPECIALIZATION was beginning in American medicine a hundred years ago; the ancient schism between surgeons and those who relied on medicines became much involved as men elected to follow special lines of investigation. All, however, began with similar training: two years of identical lecture courses, followed by preceptorships under leaders of varying enthusiasms. For a generation after the Mexican War, otolaryngology and ophthalmology grew up together, and the "eye-ear-nose-throat specialist" secured such knowledge as he might from leaders in Boston, New York or Philadelphia who had themselves sought experience in the clinics of Paris, France, or London, England.

Interrupted by the war between the states, development of teaching centers for otolaryngology was relatively rapid after 1866. Ether anesthesia, started at Boston in 1846, made extensive operative procedures possible, but antiseptic and aseptic technic were far in the future. Hence mastoid disease often was permitted to go on to "brain fever," unless sufficient local signs were present to suggest the simple retroauricular incision of Wilde. Not until after 1868 was trephining of the mastoid process practiced, and the mastoid cell structure often became invaded by reason of forcible irrigation through the trephine opening in an effort to "wash out" the infectious process through an existing hole in the drum membrane or down the eustachian tube into the throat. Ears were allowed to "break," often with the assistance of hot poultices. Bougenage of the eustachian tube, done to improve hearing, was recognized as a possible cause of acute otitis media.

The first case report in the first volume of the *Transactions of the American Medical Association,* issued in 1848, is an illustrated account of the operation and instruments for scarification of the upper aspect of the larynx for edema of the glottis as performed in 1846 by Gurdon Buck, a general surgeon of New York. He was later known as the father of laryngeal surgery in America. Similarly, Horace Green is acclaimed as the pioneer American laryngologist because he insisted in 1846 that he was able by swab and syringe to introduce medicaments—

Prepared at the request of the Council, Section on Laryngology, Rhinology and Otology, for the Centennial Session of the American Medical Association to be held in Atlantic City in June 1947.

chiefly solutions of silver nitrate—into the larynx between the vocal cords. Both these men worked by touch, not by sight. The laryngoscopic mirror was discovered ten years later by Manuel Garcia, a London singing teacher, and Louis Elsberg, of New York, described instruments for intralaryngeal treatment and examination in the *Transactions of the American Medical Association* for 1863. Sponges and camel's hair brushes affixed to curved laryngeal applicators were in general use as late as 1895.

The nasal accessory sinuses were terra incognita for a generation after 1846; while their bony structure was identified, little attention was paid to their ostiums or their linings, and their physiology and pathology were not worked out until recent years. Nasal treatment in the old days was limited to the splinting of fractures, the avulsion of polyps (described by McRuer in 1850) and, after 1880, to various schemes for lessening nasal obstruction due to septal deviation, such as Asch's refracture and perforated splints.

Although Physick had devised a tonsillotome in 1825, surgeons were afraid of hemorrhage during or after tonsillar operations and were prone to resort to ligation of the base of the tonsil with heavy catgut or silver wire. The slicing tonsillotome of Mackenzie came into use during the seventies, and was not displaced by the snare, by scissors or by the La Force and Sluder tonsillectomes until this century. While adenoids were noted by Meyer, of Copenhagen, in the sixties, American otolaryngologists paid little attention to them for many years.

Foreign bodies in the air and food passages were sought blindly with the finger or with various sorts of expansible bristle probangs or tilting coin catchers with flexible whalebone shafts. Mussey, in 1850 *Transactions of the American Medical Association,* reported removal of a cocklebur from the larynx by laryngofissure and upward pressure with his finger through the wound. Investigation of the upper part of the esophagus with a water-cooled speculum lighted by a white-hot platinum coil was attempted in New York in 1880; but this proved dangerous, and such speculums had to await development of small electric lamps after 1885.

Interesting medical and surgical cases were collected into yearly progress reports at the meetings of the American Medical Association and published in the *Transactions*; these cases were gleaned from local American medical journals and from foreign sources.

As the tendency toward specialization developed, various sections were formed within the Association. However, the joint Section on Ophthalmology and Otolaryngology was not authorized until the Buffalo meeting of 1878, and the Section first met under the chairmanship of Hermann Knapp, of New York, in Atlanta at the meeting of 1879. The Section on Otology, Rhinology and Laryngology received independent status in 1888, its membership being open to all members of the American Medical Association.

The American Ophthalmological and Otological Society was formed in 1868, mainly among specialists of Boston, New York and Philadelphia, and by 1871 those interested only in the ear withdrew at Providence, R. I., to form the American Otological Society. The American Laryngological Association was formed in 1879 in New York. It should be remembered that these small American societies, limited in membership, antedated all similar groups in England and on the continent of Europe. The American Laryngological, Rhinological and Otological Society was founded in New York in 1895, to reach a somewhat larger group of men, who qualify for membership by submitting theses. The American Academy of Ophthalmology and Otolaryngology began as a western group, in Kansas City in 1896. However, its policy of round table luncheon conferences developed into intensive teaching courses, and its requirement for membership of certification by the respective examining boards have brought it the largest and most influential nationwide membership. The Pacific Coast Oto-Ophthalmological Society was similarly organized in San Francisco in 1916; at present many state and regional groups are engaged in the promotion of "refresher courses" and "clinical conferences," as well as in their regular meetings.

In spite of their lack of state-supported special hospitals and pathologic institutes (which had been richly endowed in Germany and Austria following the lucrative Prussian victories of 1866 and 1871) American otolaryngologists discussed, debated and drew great profit from the cellular pathology of Virchow, the experimental methods of Claude Bernard and the antiseptic theories of Tyndall, Lister and Pasteur. American practitioners of the seventies and early eighties added to their clinical experience with Morell Mackenzie and Lennox Browne in London, or studied pathology with Virchow and bacteriology with Koch in Berlin, or discussed otology with Politzer in Vienna.

At home, such institutions as the Massachusetts Eye and Ear Infirmary (founded in 1828), the New York Eye and Ear Infirmary (founded in 1822), the Manhattan Eye and Ear Infirmary, the Philadelphia Policlinic and Graduate Hospital, the Chicago, later Illinois Eye and Ear Infirmary (founded in 1858), the New Orleans Touro Infirmary (founded in 1876) and numerous private clinics furnished students with increasing opportunities. H. P. Mosher founded the first postgraduate course at Harvard in 1898.

Early leaders in the development of otolaryngology as a specialty during its early years included the founders of these institutions and other great teachers, among them in laryngology Horace Green, Elsberg, Solis Cohen, Lefferts, Delavan, Mayer, Mackenzie; in otology, Agnew, St. John Roosa, Blake, Green, Bosworth, Deuch, Pierce, Duel; in rhinology, Asch, Freer, Ballenger, Mosher, Sluder, Skillern; in broncho-esophagology, Jackson, Mosher, Lynch, Lynah; and in forwarding

special training away from eastern centers, E. L. Shurly, Detroit; John Green Sr., St. Louis; E. Fletcher Ingals and J. C. Beck, Chicago; Arthur de Roaldes, New Orleans; Henry Wagner and Adolph Barkan, San Francisco; John C. Barnhill, Indianapolis; Robert Levy, Denver; Samuel Iglauer, Cincinnati; H. C. Birkett, Montreal; Perry Goldsmith, Toronto, and many others.

Undergraduate instruction in otolaryngology sixty years ago was virtually nonexistent; it developed slowly in the limited and primitive outpatient facilities of those days, with argand gas burners and a few borrowed head mirrors. Clinical lectures made little impression on students busy with general medicine, obstetrics and surgery. Nowadays, although time is still limited, excellent undergraduate textbooks are available, and instruction in routine examination of the ear, nose and throat is carried out in well equipped clinics. Many students provide themselves with battery-lighted ear and nasal speculums for bedside use. Among the outstanding pioneers in the improvement of undergraduate instruction have been H. P. Mosher, Harvard; C. G. Coakley, Bellevue; George Fetterolf, George M. Coates, and George Wood, Pennsylvania; Fielding Lewis, Jefferson; S. H. Crowe, Johns Hopkins; R. C. Lynch, Tulane; L. W. Dean, Iowa; G. Sluder, Washington; H. W. Loeb, St. Louis; J. F. Barnhill, Indianapolis; George Shambaugh Sr., Rush; W. E. Casselberry and J. Gordon Wilson, Northwestern; Horace Newhart, Minnesota; E. C. Sewall, Stanford. Many other gifted teachers are still carrying on this important work. Residencies in such medical school clinics and hospitals have become a most valuable factor in present day training of the otolaryngologist.

Physicians who wanted to get instruction in otolaryngology in the late eighties and early nineties caused a considerable demand for brief preparatory courses—six weeks to four months. In certain instances, these courses gave only the most superficial acquaintance with the specialty. Instruction in special pathology was notably lacking. Similar short courses were devised by English-speaking teachers abroad, notably in Vienna, Austria and Berlin, Germany, so that half-trained persons who had learned nothing but a few operative technics were privileged to return to their homes for a lucrative career as "specialists." Time showed that many capable physicians came home from these courses only to find in emergencies how poor their basic training had been; such men kept on with their studies, and returned to seek further instruction here and abroad. Nevertheless, many inferior men felt themselves highly qualified and were so regarded in their own communities.

Deficiencies in training became manifest early in the first World War at the Fort Oglethorpe training center for army otolaryngologists and ophthalmologists. While attempts were made to utilize such imperfectly qualified personnel, serious difficulties arose in Army camps in this

country and in Army hospitals abroad. The system of consultants established by the Surgeon General was effective, especially in Europe, in standardizing methods of practice and minimizing unnecessary surgical procedures.

However, it had become evident to leaders in the national ophthalmologic and otolaryngologic societies that improvement in education for the practice of these specialties was imperative. For this reason, examining boards were established in 1921 for eye and in 1924 for ear, nose and throat specialists, providing for certification of voluntary applicants. The American Academy of Ophthalmology and Otolaryngology and later each of the other national otolaryngologic societies appointed examiners and furnished initial funds for these examinations, and these societies have ever since required that the applicant possess the certificate of the appropriate board before being approved for election to membership. The Board of Otolaryngology, first headed by George E. Shambaugh Sr., with Hanau W. Loeb as secretary, has for many years been under the presidency of H. P. Mosher, with Burt R. Shurly as vice president. Among its original members are J. C. Beck, R. C. Lynch, R. H. Skillern, T. E. Carmody, T. H. Halsted, Thomas J. Harris, W. P. Wherry and Frank R. Spencer.

Following the example of the otolaryngologists and ophthalmologists, physicians in other specialties set up similar examining boards, and after due consideration the Council on Medical Education and Hospitals of the American Medical Association in 1933 approved this method of establishing the special qualifications of certain practitioners and set up general principles governing the amount of time which must be spent in special study, requirements for residencies and the like. An advisory board for the medical specialties now assists in this coordination. It was found that many so-called "basic" courses were lamentably deficient in certain subjects, notably special pathology and physiology. The institutions affected have all made notable curricular improvements in the past ten years.

These improvements in special teaching became manifest during World War II, in which possession of a certificate from the Board of Otolaryngology secured better recognition for the civilian physician entering military service. As many certified physicians as possible have been placed on the staffs of hospitals of the United States Veterans Administration, for the supervision of residents who may be eventually qualified to appear for the examinations of the various boards.

Great improvement in the level of information among American otolaryngologists followed the establishment of special journals; case reports and new procedures were no longer buried in local journals and reinterred in the "progress reports" of general medical societies. The first special journal was the *Archives of Ophthalmology and Otolaryn-*

gology, established in 1869 by Hermann Knapp, of New York, who dropped the otologic papers after 1880. The *Archives of Laryngology* was issued, 1880 to 1884, by Elsberg and others in New York. J. P. Parker, of St. Louis, founded the quarterly *Annals of Ophthalmology and Otolaryngology* in 1892. In 1896 he started a separate ophthalmic journal, and H. W. Loeb, succeeded by L. W. Dean and A. W. Proetz, continued the quarterly *Annals of Otolaryngology* until the present time. The *Laryngoscope* was founded in St. Louis by M. A. Goldstein in 1896; since his death it continues with T. E. Walsh as editor. The ARCHIVES OF OTOLARYNGOLOGY was established in 1925 by the Trustees of the American Medical Association in Chicago, with George E. Shambaugh Sr. as chief editor, succeeded after a few years by George M. Coates. Other small privately owned journals have risen and fallen with the years. The transactions of each of the national otolaryngologic societies have become available in the libraries of medical schools and metropolitan medical societies throughout the nation. International abstracts, long found but once a year in the transactions of general medical societies, are now found in current issues of national otolaryngologic periodicals.

Good-sized special libraries are to be found in some of the medical schools, like the Barkan collection in the Lane Library at San Francisco and also those in the New York Academy of Medicine, the College of Physicians of Philadelphia, the John Crerar Library of Chicago and the Army Medical Library in Washington, D. C.

The development of American otolaryngology in the past twenty years has reflected the vast achievements in all fields of general medicine and surgery. In otologic surgery, the irrigations with dilute solution of sodium hypochlorite (Dakin's solution) and the chlorinated oils of World War I gave place in the last few years to the general and local use of the sulfonamide compounds, and more recently to penicillin and the other antibiotics. Surgical mastoiditis has been cut down by early use of penicillin, after appropriate early myringotomy. Intracranial and circulatory complications of mastoid disease have been greatly reduced and radical mastoidectomy is becoming relatively infrequent. Safe routes for reaching suppuration in the petrous tip have been devised, although such deep infections are now usually controlled by antibiotics. Facial palsy, especially that of traumatic origin, is no longer repaired by hypoglossal or glossopharyngeal splicing but is readily amenable to decompression of the nerve in its canal and, if necessary, to filling in the gap with a nerve graft.

Mapping of the routes by which pain is referred to the ear, the occiput and the shoulder has been accurately done by experimental methods and includes those myalgias due to localized ischemia of sympathetic origin.

Revival and perfection of the technic for fenestration of the vestibular bony labyrinth in cases of clinical otosclerosis produced many spectacular improvements in hearing. Tinnitus has been attacked by removal of the tympanic neural plexus over the promontory as well as by section of the eighth nerve at the internal auditory meatus. Vertigo has been cured by cautery destruction of the semicircular canals.

While aviation medicine was in 1917 largely based on vestibular reactions, experience with civilian aeronautical tests demonstrated that depth perception and visual acuity were more important to the pilot. Of course, irritable labyrinths caused rejection, and normal hearing was required; but it was found that the noise of airplane engines in time became a distinct occupational hazard. Hence audiometric tests of hearing, as well as determination of altered conditions in the middle ear, eustachian tube and accessory sinuses during changes of altitude, became problems for the Army and Navy schools of aviation medicine before the recent world war. The civilian otolaryngologist is not infrequently called on in cases of aero-otitis among plane passengers unused to altitude flying.

Audiometry by electronic instruments has in the past twenty years become increasingly valuable for recording tone losses, for deciding on the type of deafness which may be present and for making permanent military and medicolegal records. Hearing aids have been greatly improved by the use of smaller radio tubes. Mapping of tonal areas in the temporosphenoid cortex with reference to corresponding cochlear areas has been accomplished by the use of electronic action currents. Effects of noise, blast and high or low air pressure have been carefully studied, experimentally and on military personnel.

The American Hearing Society, founded as the League for the Hard of Hearing in 1919, has done much to lighten the lot of deafened persons. An excellent program of rehabilitation for deafened veterans is already well established. Public school testing now picks out children who are becoming deaf, and, through cooperation with otologists, treatment by removal of adenoids or by irradiation of peritubal lymphoid masses is effected.

Radical and often excessive surgical treatment of sinuses, advocated in the early nineties and revived with improved technics in the recent twenties, has given place to semiconservatism because of increased understanding of nasal anatomy and physiology. Differential diagnosis of allergy has excluded numerous congestive processes formerly brought to operation. Turbinates are now preserved whenever possible; their reduction by electric cautery is no longer done, while diathermic coagulation is also infrequent, being reserved for control of hemorrhage. The local application of fibrin foam, cellulose or gelatin and the parenteral injection of calcium salts and vitamin K have recently been added

to time-honored pressure and packing methods of hemostasis. Routine examination of the blood, including a platelet count and the determination of the bleeding and clotting times, has been generally utilized by otolaryngologists only within the past dozen years. Submucous septal resection, high and complete, has been found to unlock crowded ethmosphenoid areas. Improved local anesthesia has made extensive nasal plastic and reparative maxillofacial procedures possible.

Controversy over partial or total tonsillectomy raged in the nineties, but has generally been resolved in favor of total removal, if scarring of the faucial muscles can be avoided. Retropharyngeal abscesses in children are opened with the head low to avoid inhalation; and this head-low recumbent position during tonsillectomy and adenoidectomy has almost done away with abscess of the lung and atelectasis from inspired septic material. In adults, lateral pharyngotomy has become the route of choice for deep pharyngeal infections.

The generation of laryngologists who became expert in the management of indirect instruments as seen in the mirror is rapidly passing away. Electric-lighted pharyngoscopes are now used by anesthetists to pass tubes for intratracheal anesthesia. Almost all laryngeal surgical procedures are carried on through direct, lighted speculums, and in a few clinics, by suspension laryngoscopy. Use of the laryngeal mirror is taught to undergraduates, who are encouraged to its diagnostic use, but too often its accurate employment is shirked by those who depend on "direct" laryngoscopy alone. Accurate diagnosis of the character of laryngeal growths is now commonly made through biopsy preceding further surgical measures, which should not be delayed. Many growths which do not fix a cord are now removed by laryngofissure, often without tracheotomy, or by thyrotomy involving one or both alae, with tracheotomy. Complete destruction of the suspected area by diathermy after surgical removal of a growth is advised. Laryngectomy is the operation of choice for extensive intrinsic cancers, but when both arytenoids are fixed or when invasion of the upper part of the esophagus has occurred, and also when glands are palpable in the neck, fractional irradiation by high voltage roentgen rays or radium pack is advised.

Nowhere has American medicine displayed more originality than in its development of distally lighted tubes and speculums and of forceps, safety pin closers, diathermic coagulation devices, esophageal sounds, bougies and dilators, batteries, suction apparatus and other incidentals to modern work in the larynx, tracheobronchial tree and esophagus. Beginning with emphasis on removal of foreign bodies, it was early found that European types of proximally lighted instruments gave inferior results. Surgical removal of laryngeal growths; cauterization of tuberculomas of the larynx and epiglottis; diagnosis and relief of

atelectasis and pulmonary abscess; biopsy and implantation of radon seeds for peribronchial cancer; relief of low esophageal spasm and fibrosis; coagulative injection of varicosities of the lower part of the esophagus; gradual dilation of strictures due to lye burn—all these are assisted by accurate roentgenography and more recently by tomographic series of films.

General use of diphtheria antitoxin and toxoid immunization have done away with O'Dwyer's intubation tubes, lifesaving device of the eighties; but tracheotomy is still a vitally important factor in saving neglected patients.

Otolaryngologists with long experience have been training thoracic surgeons and gastroenterologists in the use of appropriate instruments for diagnosis in their fields. Many hospitals for patients with tuberculosis now require their surgical residents to be grounded in bronchoscopy.

Local anesthesia, made possible by the introduction of cocaine in 1884, was greatly improved by the isolation of epinephrine in 1901 and the synthesis of procaine hydrochloride, tetracaine hydrochloride and other agents, safer for surface or hypodermic use than cocaine, during more recent years. The cocaine spray is now rarely employed and never prescribed in otolaryngologic practice. Along with other highly flavored and colored solutions, it was commonly seen when compressed air spraying became popular in the early nineties. More recently, anesthesia obtained by direct application to nerve trunks in the nose, or by submucosal injection in the pharynx, or by subcutaneous nerve block about the neck, external nose, forehead, orbit, mastoid and concha, has permitted extensive operative procedures which formerly required long and sometimes dangerous subjection to nitrous oxide–ether anesthesia. Of course, patients who are to undergo such operations as tympanomastoidectomy, laryngofissure, external frontal sinusotomy and transorbital or transantral ethmoidectomy must all have strong preoperative sedation before local anesthetization can be successful.

Introduction of liquid medicinal agents into the trachea, for many years managed by direct syringing through a curved cannula, was facilitated through spray vaporization. However, liquid petrolatum sprays and nose drops have within recent years been found unabsorbable in the alveoli and occasionally productive of fibrotic changes. Contrast mediums—vegetable oils containing iodine, introduced to obtain roentgenographic visualization of the tracheobronchial tree—have been found to remain in the lungs for many months without apparent harmful effects.

While penicillin has been found valuable when forcibly inhaled into the lungs from oxygen or helium cylinders, recent experience has demonstrated that ordinary atomizer spraying of fresh penicillin dilutions in isotonic solution of sodium chloride is just as effective.

Collaboration with ophthalmologists has clarified opinion regarding the debatable effects of posterior sinus disease or retrobulbar optic neuritis, and the differential diagnosis of orbital or ocular neoplasms from mucoceles or low grade ethmoiditis. Ocular headaches resulting from refractive error or incipient glaucoma have been clearly distinguished from those due to sinus involvement. The relation of latent sinus disease to low grade recurrent iridocyclitis and glaucoma has been well established. Danger to ocular structures from necessary irradiation of deep-seated nasal, antral or nasopharyngeal cancers has been thoroughly recognized.

Collaboration in the large national societies, especially the American Academy of Ophthalmology and Otolaryngology, has led to the establishment of the National Registers of Ophthalmic and Otolaryngic Pathology in the Army Medical Museum, the publication of atlases of these subjects, and the provision of study sets of microscopic slides for the use of candidates for board examinations, residents and interns. Pan-American congresses, inviting accredited specialists from Latin America and the Antilles, have also been sponsored by the American Academy, and many students of otolaryngology from these countries have, during recent years, been receiving their preparation from institutions in the United States.

American anatomists have for years devoted special study to the problems of otolaryngology, notably at Harvard, Pennsylvania, Johns Hopkins, Northwestern, Wisconsin, Washington, Stanford and Oregon Universities. From their basic research into neuroanatomy, embryology and histopathology, often inspired by clinicians and sustained by grants from such societies as the American Academy of Ophthalmology and Otolaryngology, have come discoveries which continue to bring light into the remaining dark corners of this special field. It should not be forgotten, however, that the curiosity and abiding interest of the American otolaryngologist, often thrown on his own resources in an isolated community, have created a powerful demand not only for vast development of technical skills but for improvement in special medical education, in periodical literature and in fundamental research.

Necessary to fulfilment of such demands is the establishment of more and better special hospitals, where adequate training in otolaryngology may be given. American otolaryngology in the past hundred years has achieved great progress; it should go on to greater things in the years to come.

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NEUROGENIC TUMORS OF NOSE AND THROAT

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NEUROGENIC tumors occurring primarily in the nose and the throat are rare, and there are probably few other tumors about which there is so much confusion in the literature. When a neurogenic tumor is encountered, and this is usually unexpectedly, one may be at a loss to know what to do, even though most of them are amenable to successful surgical correction or removal.

Any tumor derived from nerve tissue or its coverings may be considered a neurogenic tumor. Neurogenic tumors of the nose or the throat may be divided into two large groups consisting of, first, developmental errors and, second, neoplasms.

The developmental errors are meningocele, encephalocele and hydroencephalocele. In the literature these have been called sincipital [1] or basal hernias,[2] encephaloma,[3] rhinoencephalocele,[4] glioma,[5] fibroglioma [6] and neurocytoma.[7] They are most frequently referred to as meningocele and encephalocele or sincipital or basal hernias.

These three conditions have in common a defect of the bones through which some part of the cranial contents protrudes. The only exception to this is the small encephalocele; frequently this is not connected to the brain by any stalk, nor is there any evidence of a defect of the bones.

From the Section on Laryngology, Oral and Plastic Surgery, Mayo Clinic.
Read at the meeting of the American Laryngological Association, Chicago, May 29, 1946.

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1. von Meyer, E., cited by Browder and de Veer.[4]
2. Heinecke, W., cited by Fenger.[15]
3. Berger, P., cited by Browder and de Veer.[4]
4. Browder, J., and de Veer, J. A.: Rhino-Encephalocele, Arch. Path. **18**:646-659 (Nov.) 1934.
5. Jackson, C., and Coates, G. M.: The Nose, Throat, and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929, p. 166.
6. Rocher, H. L., and Anglade, cited by Browder and de Veer.[4]
7. Eggston, A. A.: Histopathology of the Nose and Throat, in Nelson's New Loose-Leaf Surgery, New York, Thos. Nelson & Sons, 1942, chap. 8, pp. 654-655.

In meningocele there is a protrusion of the membranes alone. These form a sac which communicates with the subarachnoid space and which is usually distended with fluid.

In encephalocele there is a protrusion of a portion of the brain substance or tissue resembling brain substance. This is connected with the rest of the brain by a constricted neck or pedicle except for the small encephalocele noted previously. The rarity of encephalocele is stressed by the fact that Browder and de Veer [4] have gathered from the literature data on only about 50 cases in which tissue more or less resembling brain tissue has been found subcutaneously at the base of the nose, in the nasal or nasopharyngeal cavity or in both locations.

Hydroencephalocele is a protrusion of a portion of the brain substance which contains within it part of the cerebral ventricles. It may or may not be congenital. If it is acquired, it appears shortly after birth and is the result of hydrocephalus. The increasing intracranial pressure finds a weak spot in one of the suture lines, which yields. Herniation of cerebral contents results.

These congenital errors are most easily understood if one considers them in a general way as sincipital or basal hernias with the supposition that there is a defect of the bones or that there was a defect during embryologic development which permitted brain matter or its coverings to protrude or to be pinched off and left behind as a vestigial structure. Lampert [8] stated that the frontal and occipital portions of the embryologic medullary tube close much later than the rest of the tube. Consequently, there are greater chances for abnormalities to take place in these regions. Following this line of thought, one may assume that a sincipital hernia could produce a tumor visible externally in the midline at the root of the nose, or at the junction of the bony and cartilaginous portions of the nose, or near the inner canthus, depending on whether the actual or hypothetic bony defect was located between the nasal and frontal bones, between the frontal, nasal and ethmoid bones or between the frontal, nasal and lacrimal bones.[1] A basal hernia would not produce a tumor visible on the face. The tumor would be present in the nasal or the nasopharyngeal cavity, or even in the mouth, the posterior portion of the orbit or the sphenomaxillary fossa, depending on whether the defect was located between the sphenoid and ethmoid bones, at the spheno-orbital fissure or at the infraorbital fissure.[2]

Microscopically, the neurogenic tumors of developmental origin vary from the ordinary meningocele, consisting of a sac lined with epithelium and with a fibrous wall, to a solid tumor of the encephalocele variety, consisting of tangled masses of glial cells interspersed among strands of fibrous tissue. Occasionally atypical nerve cells may be seen.

8. Lampert, F. M.: Pathogenesis and Treatment of So-Called Congenital Cerebral Herniae, Surg., Gynec. & Obst. **38**:159-162 (Feb.) 1924.

The neoplasms of neurogenic origin encountered in the nose and throat are neurofibroma, ganglioneuroma and meningioma. In the literature, neurofibroma has been called perineural fibroblastoma, neurinoma, perineural fibroma, schwannoma and neurogenic fibroma.[9] Ganglioneuroma has been called ganglion cell neuroma and ganglioma. Meningioma has been called dural endothelioma, psammoma, neuroepithelioma and meningeal fibroblastoma.

Neurofibroma arises from the specialized investment which separates and insulates nerve tissue from surrounding structures. It is usually recognized as a fibroblastoma arising from the connective tissue sheath (perineurium) of a nerve. Some believe that it arises from the cells of the sheath of Schwann (schwannoma) and is therefore ectodermal in origin.[9] It forms a benign encapsulated tumor of the sheath of a spinal nerve root, a cranial nerve or a peripheral nerve and appears as a solitary tumor, much more often in a central location [10] than in a peripheral one. Occasionally neurofibroma is multiple, with hundreds of tumors growing from the fine cutaneous nerves, deeper and visceral nerves and cranial nerves—a condition known as von Recklinghausen's disease. It is not uncommon to find a neurofibroma in the nose or throat of a patient with this disease.

Microscopically, neurofibroma is composed of long slender cells, the elongated nuclei of which are arranged in palisades grouped into eddies or streams. Sometimes there is a great deal of myxomatous, jelly-like material present and the typical palisading streams and eddies may not be so evident. There may be hemorrhages or other evidences of degeneration, such as cyst formation, particularly in the larger tumors of the pharynx.

Ganglioneuroma is a nerve tumor containing adult ganglion cells, glial cells and nerve fibers. The most generally accepted hypothesis concerning its origin is that it arises by multiplication of embryonal neurocytes which continue to multiply and differentiate through the various stages up to adult ganglion cells.[11] It is exceedingly rare in the nose and throat. Geschickter [12] said that portions of the olfactory ectoderm in the upper ethmoid region are neuroepithelial in character and that ganglioma may rarely form from these cells.

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9. Boyd, W.: A Textbook of Pathology: An Introduction to Medicine, ed. 3, Philadelphia, Lea & Febiger, 1938, pp. 961-964.

10. Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 968.

11. Bosse, M. D.; Nettrour, W. S.; Soffel, J. A., and Rohm, J. Z.: Ganglion Cell Tumors of the Neck: Two Case Reports, Am. J. Surg. **65**:120-122 (July) 1944.

12. Geschickter, C. F.: Tumors of the Nasal and Paranasal Cavities, Am. J. Cancer **24**:637-660 (July) 1935.

Meningioma is usually believed to be a benign tumor arising from some component of the meninges. It is generally accepted now that this tumor is derived from an embryonal rest of the arachnoid. To explain primary extracranial meningioma occurring in the paranasal sinuses or subcutaneously at the root of the nose, one might assume that embryonal arachnoid rests were pinched off and left behind in these locations during embryologic development and became neoplastic at a later date. Others might account for the presence of meningioma in these locations by assuming that some ancient trauma or cerebral hypertension displaced bits of arachnoid that potentially were neoplastic.

Microscopically, meningioma shows elongated fibroblastic nuclei in a whorled arrangement. Here and there the whorls may have undergone hyaline change, forming the typical sandlike bodies called psammomas, from the term Ψάμμος (sand).

We have not included chordoma in this group because, strictly speaking, it is not a neurogenic tumor since it arises from a remnant of the primitive notochord.

In the last twenty-seven years at the Mayo Clinic 33 cases of neurogenic tumors of the nose and the throat have been encountered. These include 9 cases of developmental errors (6 cases of meningocele and 3 of encephalocele) and 24 cases of neoplasms (19 cases of neurofibroma, 1 case of ganglioneuroma and 4 cases of meningioma).

MENINGOCELE

There were 6 cases of meningocele (fig. 1). In 5 cases the tumor was external (at the root of the nose), and in 1 case it was intranasal. In 4 cases there was a large, pulsating, compressible congenital tumor at the root of the nose. In another case there was a small, firm, rounded mass at the root of the nose, which was thought to be a dermoid cyst. At the time of the operation it was recognized as a meningocele, and the pedicle, which entered the cranium at the midline, at the junction of the frontal and nasal bones, was ligated. The child had had epileptic attacks, and the mother said that these ceased after the operation. In 1 case the tumor presented as a cyst of the nasal cavity. Rhinorrhea developed after an intranasal operation elsewhere. Intranasal meningocele appears to spring from the septum but is really arachnoid and dura protruding through the cribriform plate of the ethmoid bone to one side or the other of the crista galli.

It is hard to confuse meningocele with any other tumor. Intranasal meningocele may be difficult to identify, but any purplish cystic mass that dents with a probe and that is causing nasal obstruction in a child should be considered as meningocele, and particularly so if there is any dripping of clear cerebrospinal fluid after aspiration or attempts at removal.

External meningocele at the root of the nose is generally covered with skin but may be covered only with granulation tissue. Other deformities, such as spina bifida, clubfoot, harelip or mental deficiency, are frequently present. External meningocele may be confused with cavernous hemangioma, which does occur in this region. A roentgenogram of the skull may, and usually does, show a defect of the bones. When this defect is present it will aid in a differential diagnosis.

Meningocele is a neurosurgical problem, and we shall not discuss it further except to say that aspirating an intranasal cyst in a child is

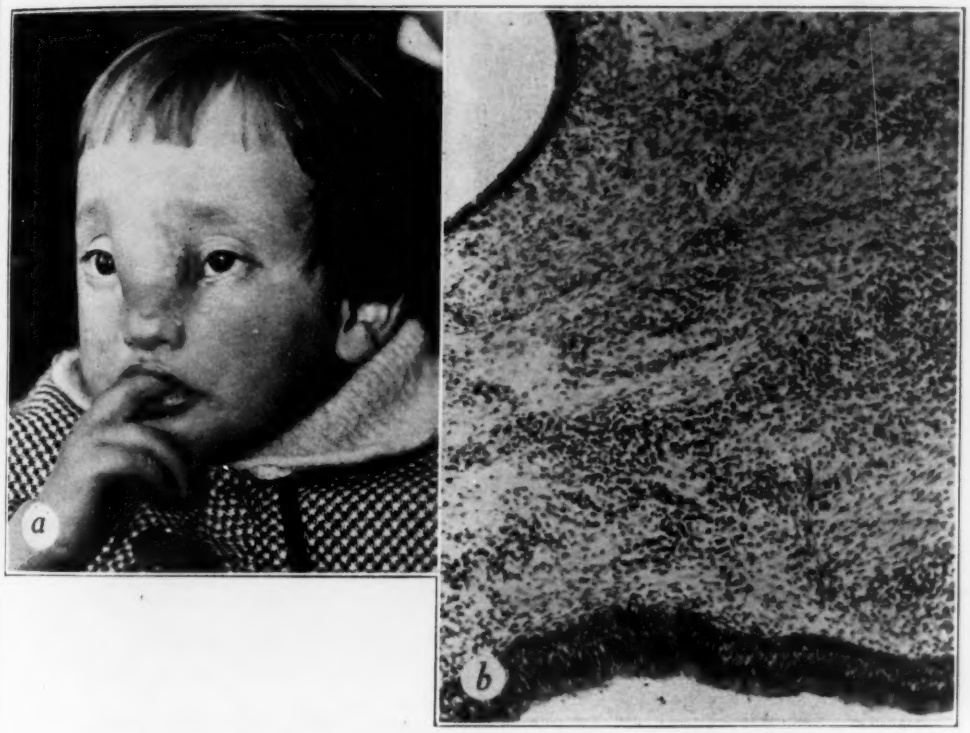

Fig. 1.—(*a*) Meningocele of the bridge of the nose. (*b*) Photomicrograph of meningocele (× 100).

hazardous because of the possibility of fatal meningitis following such a procedure.

ENCEPHALOCELE

There were 3 cases of encephalocele (fig. 2). In each case the tumor was external (at the root of the nose), lying just to the right of the midline. The three tumors seen by us were firm, rubbery, round congenital tumors. There had been practically no change in size since birth, and the average diameter was 1.3 cm. They were mistaken pre-

operatively for dermoid cysts. In a case reported in the literature an encephalocele was mistaken for a mixed tumor because of the firm, rubbery consistency.[13]

There was no intranasal extension of any encephalocele seen by us, but such extensions have been described in the literature.[14] In some instances the tumor may extend all the way to the brain, as in Lampert's case. It is usually impossible to remove such a tumor without the death of the patient during the postoperative period. In a few cases meningitis and death have followed the extirpation of a small encephalocele which apparently had no gross intracranial connection. In Eggston's [7] opinion this result is due to the presence of a sheath similar to that surrounding

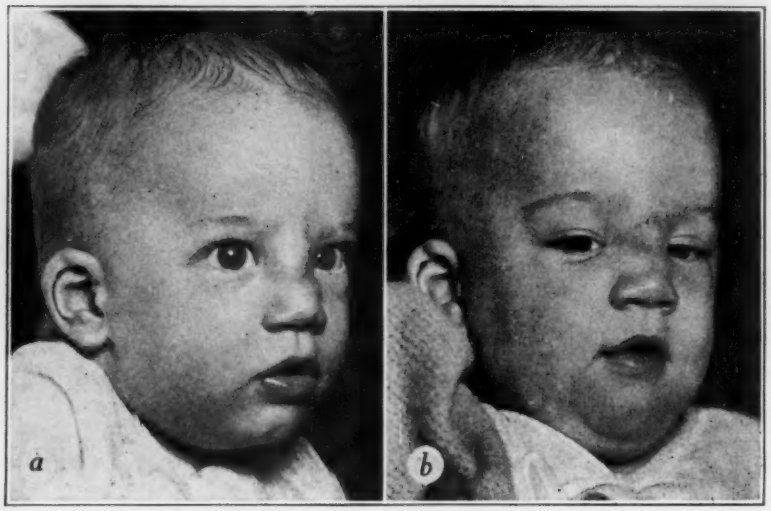

Fig. 2.—Encephalocele of the nose: (*a*) preoperative and (*b*) postoperative appearance of the patient.

the olfactory or other cranial nerve, along which infection was able to travel to the cranial tissue.

The small external encephalocele with no intranasal or intracranial connection is best removed through a horizontal incision over the root of the nose so that any scarring that results will be minimal and easily covered by a pair of spectacles. The tumor is not encapsulated and must be removed by sharp dissection, care being taken to remove all of it (fig. 3). There may be a small teatlike projection which fills a

13. Fèvre, M., and Huguenin, R.: Encéphalocèle atypique réalisant une tumeur solide à la racine du nez, Ann. d'anat. path. **13**:333-336 (March) 1936.

14. Clark, J. P.: Glioma of the Nose, Am. J. M. Sc. **129**:769-774 (May) 1905.

dehiscence between the two nasal bones or at the junction of the bony and cartilaginous portions of the nose. This was seen in 2 of our cases and has been described previously.[13]

An intranasal encephalocele may have intracranial connections. For this reason, any surgical procedure undertaken for its removal must furnish adequate exposure so that the distal pedicle or attachment of the tumor may be ligated or electrocoagulated if there is any evidence of intracranial connection. Fenger [15] turned down the maxillary bone through an external incision to remove such a tumor successfully. The

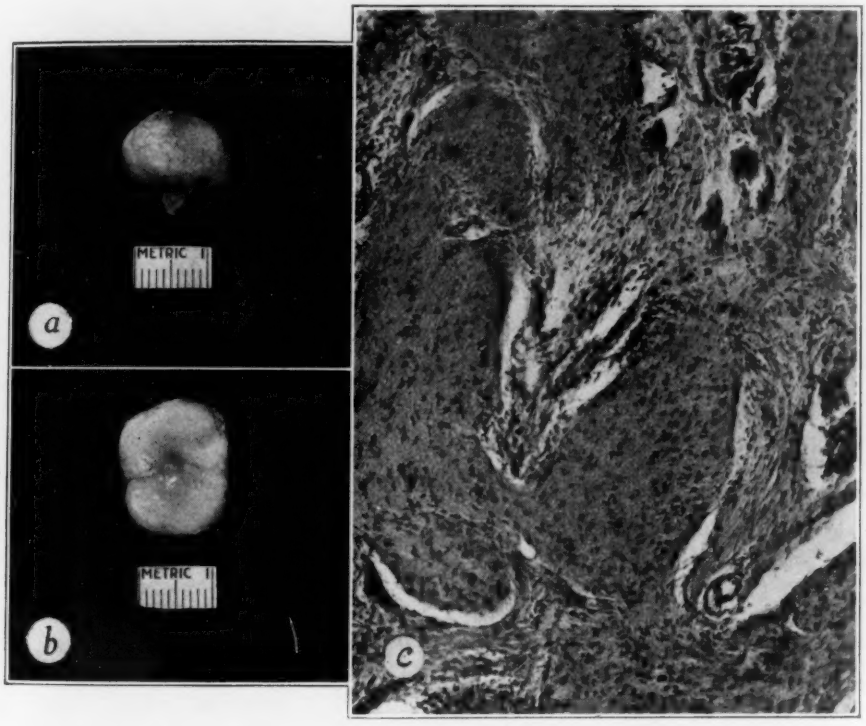

Fig. 3.—(*a*) Gross appearance of the excised encephalocele. Note the teatlike projection, which filled a dehiscence between the two nasal bones. (*b*) Gross specimen on section. (*c*) Photomicrograph of encephalocele (× 100).

transantral approach for an intranasal or an antral tumor advocated by New and Erich [16] or the frontoethmoid approach would also furnish adequate exposure.

15. Fenger, C.: Basal Hernias of the Brain, Am. J. M. Sc. **109**:1-17 (Jan.) 1895.

16. New, G. B., and Erich, J. B., in Jackson, C., and Jackson, C. L.: Diseases of the Nose, Throat, and Ear, Philadelphia, W. B. Saunders Company, 1945, pp. 72-81.

If a child has a firm, hard external tumor at the root of the nose with evidence of an intranasal tumor as well, he is almost certain to have an encephalocele. Our 3 patients had no other evidence of abnormalities or of mental deficiency. Most reports in the literature agree with this finding.

HYDROENCEPHALOCELE

There was no hydroencephalocele in our group. A typical hydroencephalocele is round and elastic, giving evidence of fluid. When the communication with the cranial cavity is sufficiently large, the tumor usually pulsates. Many of the patients exhibit signs of mental impairment or manifestations of organic brain disease. They should be in the care of a neurosurgeon, and treatment should be postponed, if possible, to determine whether there is complicating hydrocephalus.

Fig. 4.—Neurofibroma of the bridge of the nose.

NEUROFIBROMA

There were 19 tumors diagnosed as neurofibroma in our group. Their location was as follows: nasopharynx, 1; nasopharynx and hypopharynx, 1; oropharynx and hypopharynx, 3; tonsillar region, 3; larynx, 2; antrum, nasopharynx and sphenoid sinus, 1; nasal cavity, 2; vestibule of the nose, 2; bridge of the nose, 2; and ala of the nose, 2.

Of these 19 cases only 4 were cases of von Recklinghausen's disease. These included 2 in which the tumor was pharyngeal, 1 in which the nasal ala was involved in a diffuse neurofibroma of the upper lip and cheek and 1 in which the bridge of the nose and the supraorbital region were involved. In the latter 2 instances the neurofibroma was a special kind, commonly seen on the face (fig. 4), in the distribution of one or more of the branches of the fifth cranial nerve, in patients with von Recklinghausen's disease or in those who have some stigmas of the disease. Clinically neurofibroma is frequently mistaken for angioma

because of its sponginess and diffuseness, especially if the patient has only a few of the typical pigmented spots on the trunk or only a few small inconspicuous tumors on unexposed parts. They are instantly recognized once they are cut into at operation. Neurofibroma is composed of loosely assembled masses of small individual growths varying from 2.5 mm. in size, sometimes larger. Grossly it looks like a cluster of small white grapes or tiny white marbles.

Neurofibroma is a slow growing, benign tumor. The average age at the onset of symptoms was 26.9 years. The average duration of symptoms was 7.8 years. Neurofibroma may appear cystic or solid with no overlying ulceration. If it is located in the pharynx, it is usually mistaken for a mixed tumor, although desmoid or lipoma should also be considered in the differential diagnosis. The diagnosis is made easy if

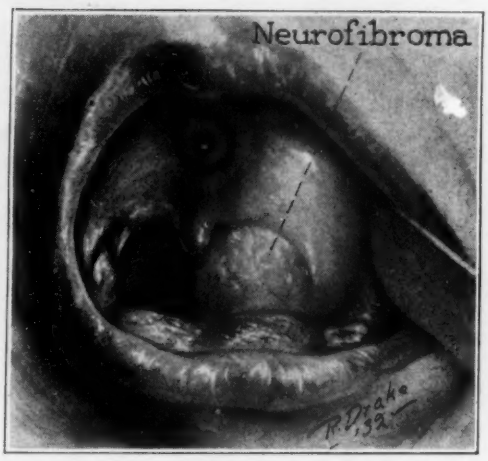

Fig. 5.—Neurofibroma of the pharynx.

there is any evidence of brownish pigmentation of the skin (café au lait) or if there are multiple tumors of the extremities or the trunk. The symptoms and chief complaint vary with the location (fig. 5). Pharyngeal neurofibroma may cause some difficulty in talking and swallowing but rarely does it reach a size large enough to produce respiratory obstruction. The patients usually have been aware of a slowly growing, painless tumor in the throat for years (fig. 6). Only 1 patient had pain and that was due to a large tumor that involved the vault of the nasopharynx. Only 1 patient complained of bleeding. The bleeding was caused by neurofibroma of the nasal fossa, primarily on one of the turbinates. The first and usually the only symptom of neurofibroma occurring primarily in the nose or the antrum is obstruction.

The only symptom in a case of neurofibroma (fig. 7) of the free border of a vocal cord was persistent hoarseness slowly increasing over

a period of ten to twelve years. In the other case of neurofibroma involving the larynx the tumor was situated on a posterior surface of the arytenoid cartilage and caused noisy breathing for one year; the patient was a child 5 years old. Up to February 1944 only 15 cases of neurofibroma of the larynx had been reported in the literature. This total includes only 1 of our cases. According to Smith,[17] neurofibroma is most likely to occur in the early decades of life, and the usual site is the ventricular band or the aryepiglottic fold. Tumors of this type are usually smooth and round, without mucosal ulceration, and it is difficult

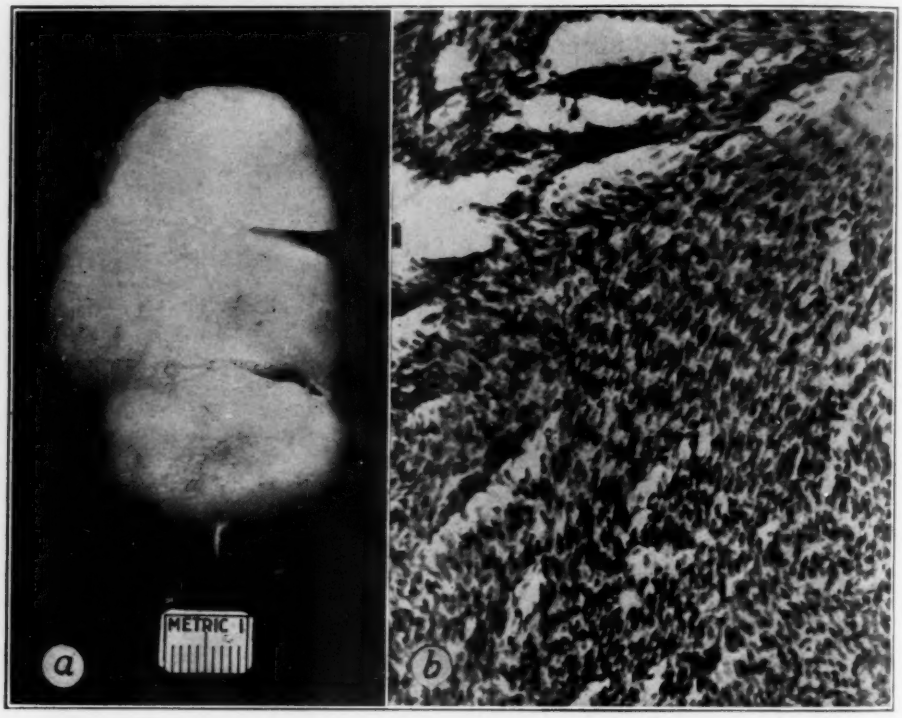

Fig. 6.—(*a*) Neurofibroma of the pharynx. (*b*) Photomicrograph of neurofibroma (× 200).

or impossible to obtain tissue for biopsy. In 1 of our cases the tumor was removed under suspension laryngoscopy, and in another it was necessary to perform thyrotomy to expose the tumor adequately and remove it.

Neurofibroma of the oropharynx, the hypopharynx or the tonsillar region is best attacked surgically through the mouth after preliminary

17. Smith, T. T.: Solitary Neurofibroma of Larynx, Arch. Otolaryng. **39**: 144-151 (Feb.) 1944.

ligation of the external carotid artery on the side involved in order to minimize bleeding at the time of removal. The growth shells out easily from the capsule and rarely recurs. An occasional pharyngeal tumor of this type may be so large that it is necessary to do preliminary tracheotomy to facilitate the maintenance of an adequate airway at all times and to minimize postoperative obstruction or the danger of fluids and secretions being aspirated into the trachea. It is usually necessary to approach the large tumor through an external incision. In 5 cases neurofibroma of the pharynx or tonsillar region was removed intraorally after preliminary ligation of the external carotid artery on the involved side. As noted by others and seen in one of our cases, there may be temporary paralysis of larynx, pharynx or palate or findings suggestive of Horner's syndrome after removal of a large neurofibroma of the pharynx.

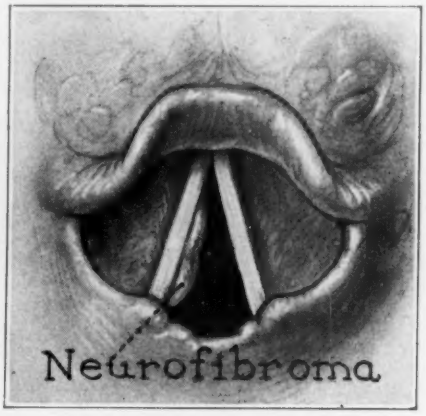

Fig. 7.—Neurofibroma of the larynx.

Sometimes, because of its location, it is impossible to shell out such a tumor. This may happen in the nasopharynx, the antrum or the nasal cavity, and consequently it may become necessary to destroy the tumor with surgical diathermy. Neurofibroma is not radiosensitive and irradiation would not be expected to produce any results.

GANGLIONEUROMA

We have encountered only 1 ganglioneuroma in the nose or the throat. This was a firm tumor involving the left basal region of the tongue of a man 26 years of age (fig. 8). It extended forward to a point opposite the bicuspid area and back almost to the tip of the epiglottis. The same tumor extended down through the floor of the mouth and presented in the left submaxillary region. It had been slowly enlarging for seven years. The patient stated that two courses of roentgen therapy had been

given, with no effect on the tumor. He came to the Mayo Clinic because of severe hemorrhages in the previous four months and at the time of examination was found to have a hemoglobin level of 6 Gm. per hundred cubic centimeters of blood. After preliminary ligation of the left external carotid artery, the tumor was removed through the mouth (fig. 9). It was a friable vascular growth surrounded by a thin capsule and had to be removed piecemeal. It measured 10 by 12 cm. and weighed 100 Gm.

Only 1 other case of ganglioneuroma of the nose or the throat has been reported in the literature. This was one of ganglioneuroma of the pharynx, reported by Foster [18] in 1942. Ganglioneuroma is an encapsulated, benign, firm, rubbery tumor usually associated with the sympathetic nervous system. Cases of ganglioneuroma of the neck have been reported by McFarland and Sappington,[19] Bosse and his associates [11] and others. It is conceivable that such a tumor might bulge into the throat and be classified as a tumor primary in that location. The surgical problems are no different from those of neurofibroma. The exact diagnosis must await microscopic examination, and this can be said for almost any tumor of the nose or the throat.

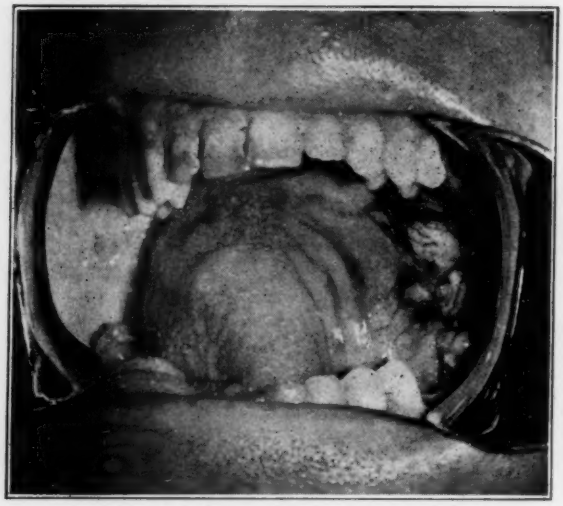

Fig. 8.—Ganglioneuroma of mouth, tongue and pharynx. Note the huge tumor on the left side.

18. Foster, J. H.: Ganglion Cell Neuroma of the Pharynx, Arch. Otolaryng. **36**:372-376 (Sept.) 1942.

19. McFarland, J., and Sappington, S. W.: A Ganglioneuroma in the Neck of a Child, Am. J. Path. **11**:429-448 (May) 1935.

MENINGIOMA

There were 4 cases of meningioma. In 3 cases the tumor was in the frontal sinus, and in 1 case it was located subcutaneously at the root of the nose.

We have not considered any case in which an intracranial meningioma, such as meningioma of the olfactory groove, the sphenoid ridge or the orbit, secondarily invaded the nose or the throat.

One of the frontal sinus tumors filled the sinus, invaded the antrum and the ethmoid sinus and encroached on the medial wall of the orbit.

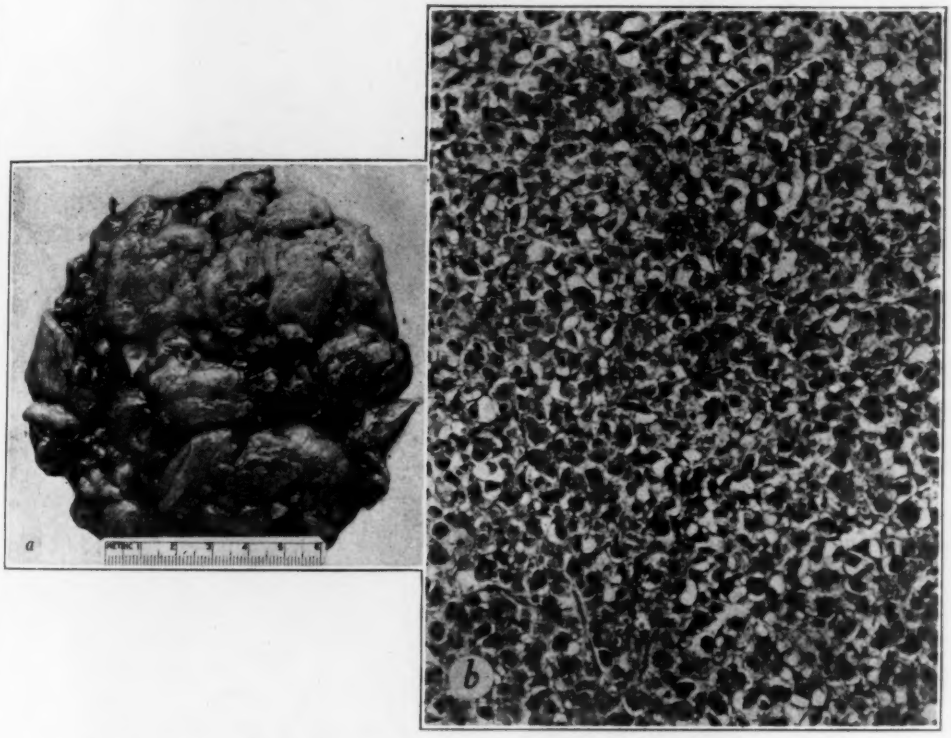

Fig. 9.—(*a*) Ganglioneuroma of mouth, tongue and pharynx (same case as fig. 8). (*b*) Photomicrograph of ganglioneuroma (× 100).

Another occurred in a woman 52 years of age, who complained chiefly of a slow-growing painless hard tumor at the root of the nose. Roentgenograms were interpreted as showing an osteoma involving the left frontal sinus (fig. 10). Surgical exploration revealed a friable vascular tumor (fig. 11) lying in the frontal sinus and attached at a few points to the inner wall.

The third was associated with a very large osteoma of the left side of the frontal bone, which caused an external deformity that had been

slowly enlarging for fifteen years. At the time of the operation, the involved bone was removed and found to contain "nests" of meningioma with a meningioma in the frontal sinus. The inner plate of the frontal bone was intact, and there was no dural attachment or evidence of intracranial meningioma. This is a good illustration of the tumor cell invasion of the bone and the hyperostosis which may develop.

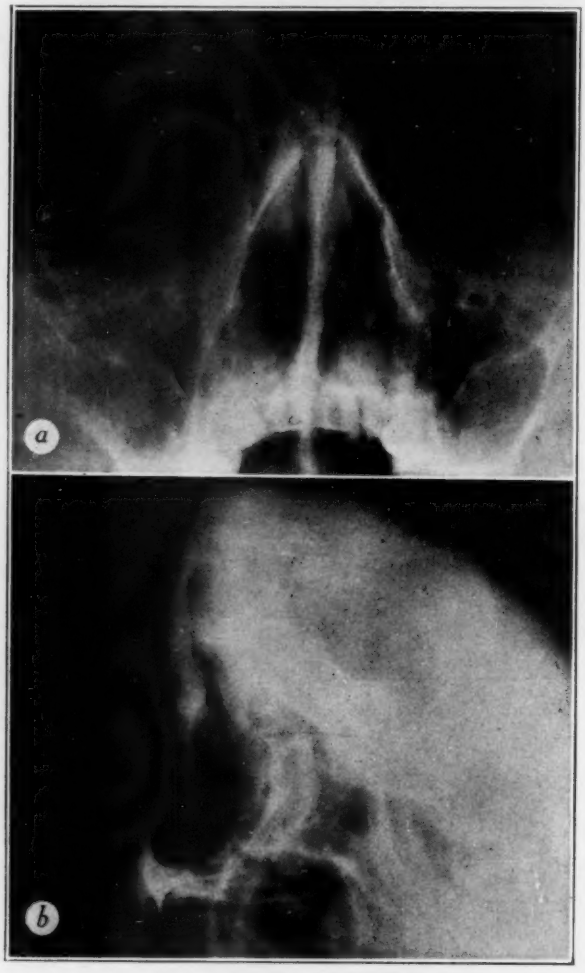

Fig. 10.—Meningioma involving the nasal bones.

The fourth meningioma, the one located subcutaneously in the midline at the root of the nose, was encountered in a woman 40 years old who had a bony-hard swelling, 2.5 by 3 cm., which had been enlarging slowly for thirteen months. A roentgenogram showed an irregular periosteal proliferation involving the nasal bones and the external surface

Fig. 11.—Meningioma (× 100).

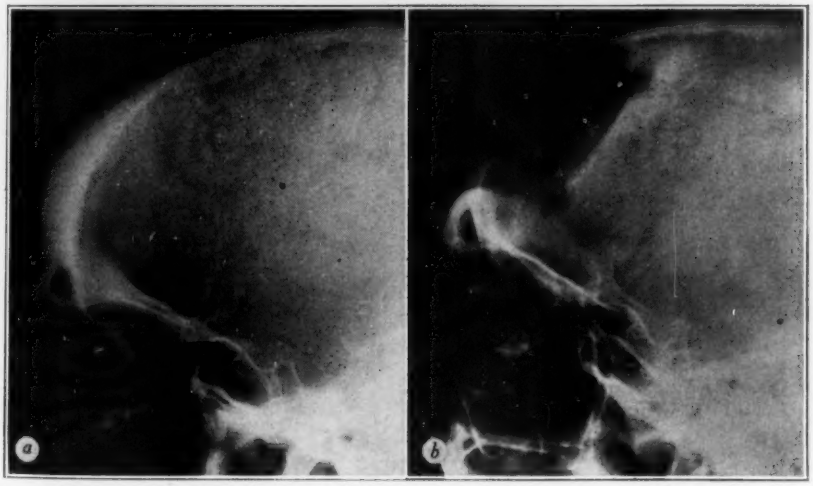

Fig. 12.—(*a*) Meningioma of the frontal bone. (*b*) Appearance after the tumor had been removed by a neurologic surgeon.

of the frontal bones. Surgical exploration through an external incision over the tumor revealed a tumor involving the base of the nasal bone and the lower part of the frontal bone. The bone had been destroyed in this region. There was no evidence of tumor in the frontal sinus, nor any intracranially or intraorbitally. The tumor was destroyed with

Fig. 13.—Casts made to show the postoperative deformity (same case as fig. 12).

Fig. 14.—Front and side view after cartilage grafts had been used to build out the defect (same case as figs. 12 and 13). It is necessary, as a rule, to make dermal grafts to smooth out the surface.

surgical diathermy, and sequestrectomy was performed two months later. Seven months and sixteen months later, small recurrences were removed with surgical diathermy. Four years later the patient wrote that she had minimal deformity with no evidence of recurrence.

Meningioma is a slowly growing expansile tumor that usually grows by pushing aside surrounding tissue. It does not metastasize but recurs locally if not completely removed. In a considerable percentage of cases it produces a marked effect on adjacent bone. There may be a slight pressure absorption or marked hyperostosis.[20] This hyperostosis may be the reaction of the bone without its having been invaded (fig. 12), but in 20 per cent of the cases the tumor tends to invade the haversian system of the overlying bone. Absorption of bone and formation of new bone occur simultaneously and may produce a large bony mass.[21] In time these tumor nests may replace the thickened bone by a core of solid tumor. The effect on adjacent bone is so characteristic that a preoperative roentgenologic diagnosis of meningioma can be made with confidence in most of the cases in which it is exhibited. This picture may be confused with chronic osteomyelitis or osteoma (figs. 13 and 14).

Meningioma occurring in the paranasal sinuses or subcutaneously at the root of the nose is best removed with surgical diathermy for two reasons: Surgical diathermy controls the bleeding which invariably occurs when one curets or dissects the tumor, and it destroys the involved bone or periosteum, preventing recurrence.

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20. Cushing, H.: The Meningiomas (Dural Endotheliomas): Their Source and Favored Seats of Origin, Brain **45**:282-316 (Oct.) 1922.

21. Brain, W. R.: Diseases of the Nervous System, ed. 2, London, Oxford University Press, 1940.

TUMORS INVOLVING THE PETROUS PYRAMID OF THE TEMPORAL BONE

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DETROIT
AND
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TUMORS involving the petrous pyramid of the temporal bone are relatively uncommon, present a wide variety of types and produce such a multiformity of subjective and objective symptoms that it was deemed advisable to report some of the more unusual cases observed at the University of Chicago Clinics in which the temporal bones were obtained for histopathologic examination. Such reports in the past have been rather infrequent, chiefly because of the tedious and complicated technics involved in preparing temporal bones for histopathologic studies.

The temporal bone itself may be a primary site of origin for a new growth. The following types of tumors have been among those reported as primary in the temporal bone: meningioma (case 1), sarcoma (case 2), squamous cell carcinoma, plasma cell myeloma, hemangioma, epidermoid and dermoid,[1] malignant melanoma,[2] cylinder cell epithelioma [3] and adenocarcinoma.[4] Jefferson and Whitehead [5] observed an interesting primary papilliferous cystoma in the petrosa consisting of a mass of closely packed cysts filled with hyaline material. They suggested that this growth may have originated from an embryonal rest because its structure indicated either a thyroid or a parathyroid origin and because there is a close developmental relationship between the thyroid gland and the middle ear. They also suggested that it

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From the Division of Otolaryngology, University of Chicago.

1. Jefferson, G., and Smalley, A. A.: Progressive Facial Palsy Produced by Intratemporal Dermoids, J. Laryng. & Otol. **53**:417, 1938.

2. Thorell, I.: Treatment of Malignant Tumours of the Middle Ear at Radiumhemmet, Stockholm, Acta radiol. **16**:242, 1935.

3. Lange, W.: Ueber die Entstehung der Mittelohr-Cholesteatome, Ztschr. f. Hals-, Nasen- u. Ohrenh. **11**:250, 1925.

4. Furstenberg, A. C.: Primary Adenocarcinoma of the Middle Ear and Mastoid, Ann. Otol., Rhin. & Laryng. **33**:677, 1924.

5. Jefferson, G., and Whitehead, R.: Papilliferous Cystoma of the Petrous Bone Associated with Hypernephroma and Cystic Pancreas, Brit. J. Surg. **19**:55, 1931.

may have originated from the anlage of the choroidal system, which is derived from the neural crest. The more common primary squamous cell carcinoma does not arise in the petrosa and will not be included in this report.

Secondly, the temporal bone may be invaded by tumors originating from contiguous areas. Such a relationship is encountered in carcinoma or lymphoepithelioma of the nasopharynx extending into the petrosa, in tumors of the parotid gland advancing into the middle ear and in tumors of the cerebellopontile angle extending into the internal auditory meatus (acoustic neurinoma, meningioma, neurofibroma [case 5], glioma [case 4], diffuse sarcomatosis of the meninges [case 3] and hemangioma). Acoustic neurinoma may arise within the internal auditory meatus and affect the petrosa early. Acoustic neurinoma is more common and will be discussed in a subsequent report.

Thirdly, the temporal bone is susceptible to being involved by metastasis of tumor cells originating from a distinct primary site such as the prostate or the mammary gland. The marrow of the petrosa is capable of filtering out tumor cells circulating in the blood stream. This process is analogous to that by which miliary tuberculous foci develop in the petrosal marrow of patients in whom tubercle bacilli are being disseminated from an active focus primary in the lung.[6] A metastatic tumor of the temporal bone was encountered in our collection—a metastasis of a lymphosarcoma primary in the cecum (case 6).

MENINGIOMA

Meningiomas originate, according to Cushing and Eisenhardt,[7] from one of the numerous cauliflower-like excrescences of the arachnoid membrane (arachnoid granulations) which lie on the outer surface of the membrane along the course of the dural venous sinuses. The arachnoid granulations are most numerous on either side of the superior sagittal sinus but may be found in many areas of the skull adjacent to the various venous sinuses. In the past these tumors have been described as sarcomas or as dural endotheliomas because of their intimate relation to the dura. These tumors actually arise from the arachnoid even though they may be firmly attached by a pedicle to the dura as well as to the adjacent bone.

The temporal bone is a potential site for meningiomas in that portion adjacent to the gasserian ganglion, in the extreme tip of the pyramid adjacent to the sella turcica, along the course of the inferior and superior petrosal sinuses and in the region of the internal auditory

6. Proctor, B., and Lindsay, J. R.: Tuberculosis of the Ear, Arch. Otolaryng. **35**:221 (Feb.) 1942.

7. Cushing, H., and Eisenhardt, L.: Meningiomas, Springfield, Ill., Charles C Thomas, Publisher, 1938.

meatus. Henschen,[8] in studying the normal eighth nerve, observed in several cases typical endothelial cell nests between the larger branches of the nerve from which pacchionian bodies (arachnoid granulations) were encountered in both internal auditory meatuses in association with a bilateral neurofibroma. Scott [9] observed the same psammoma bodies in one of his cases of bilateral neurofibroma. Occasionally meningioma may clinically be confused with neurinoma. Cushing described a solitary psammomatous meningioma arising from the anterior lip of the porus acusticus internus. Lexer [8] described a case in which primary trigeminal symptoms were associated with a tumor arising between the porus acusticus internus and the dural opening for the trigeminal root but sparing the seventh and eighth nerves; the patient recovered after extirpation of the tumor. In 1910 Grasser [10] reported 3 cases of meningioma of the temporal bone: In one a small tumor was curetted out of the tympanic cavity; in the remaining cases the neoplasm was extensive, with facial paralysis and complete deafness, and in one of these it extended through the jugular foramen into the neck, where it was palpated preoperatively.

Cushing described 5 cases of meningioma of the cavum mecklii. The typical case presents unilateral paresthesia in the trigeminal distribution, ipsilateral oculomotor paresis and slight exophthalmos. Tinnitus, deafness and a sense of fulness of the ear often occur, presumably when the growth exerts pressure on the eustachian tube. In time the tumor extends over the petrous ridge into the posterior fossa, producing headaches, choked disks, facial palsy, nerve deafness, nystagmus, instability and ataxia, dysarthria and dysphagia.

The latter disturbances are due to compression of the cerebellar peduncle and deformation of the pons with secondary hydrocephalus. These symptoms may be mistaken for those of a primary tumor of the recess or for those of bulbar paralysis.

Meningioma invading the temporal bone usually produces extensive destruction of bone without inciting regeneration of bone. Cushing reported hyperostotic bone reaction in 25 per cent of his entire series of cases of meningioma. The lack of bone reaction in the temporal bone may be influenced by the fact that the bones of the base of the skull are of endochondral origin.[11]

A notable feature of meningioma of the temporal bone is that it spares the bony labyrinth. This may be due to the fact that the osseous

8. Cited by Cushing and Eisenhardt.[7]

9. Scott, P.: Auditory Nerve Tumors, J. Laryng. & Otol. **54**:576, 1938.

10. Grasser, C.: Ueber das primäre Endothelium des Mittelohres bezw. des Felsenbeins, Ztschr. f. Ohrenh. **59**:225, 1909.

11. Bernstein, S. A.: Ueber das Verhalten des Sehädelknochens beim Duraendothelium, Virchows Arch. f. path. Anat. **290**:501, 1933.

labyrinth has a separate blood supply [12] or to the fact that the endochondral portion of the bony labyrinth is more resistant than periosteal bone to neoplastic invasion.[13] Meningioma arising within the temporal bone usually produces extensive destruction of the bone before extending through the resistant dura.

CASE 1.—C. L., a 55 year old white woman, first entered the University of Chicago Clinics, Dec. 4, 1939, complaining of severe pain in the right ear and aural discharge, both of nine years' duration. In 1930 simple mastoidectomy, and in 1932 radical mastoidectomy, had been performed without relief, and at the time of the second operation the seventh nerve had been cut.

Physical examination revealed complete paralysis of the seventh, eleventh and twelfth cranial nerves of the right side. Auditory examination showed complete nerve deafness on the right. The Wassermann and Kahn tests of the blood were negative. Studies of the blood indicated that it was normal. A spinal puncture was made on December 7. The initial pressure of the spinal fluid was 210 mm. of water; compression of the right jugular vein produced a rise to 240 mm. of water, and compression of the left jugular vein a rise to 390 mm. of water. The chlorides of the fluid amounted to 130 mg., the total nitrogen to 147 mg., the nonprotein nitrogen to 84 mg. and the total proteins to 39 mg. per hundred cubic centimeters. The cell count was 7 lymphocytes.

A revision of the radical mastoidectomy with investigation of the right lateral sinus was performed on December 14. On removal of what appeared to be fibrous tissue and granulations from the middle ear and peritubal cell region, a severe hemorrhage was encountered. This was controlled by packing. Postoperatively the patient ran a febrile course. On December 20 she became drowsy. Half of the packing in the lateral sinus was removed. Spinal dynamics showed persistence of the right lateral sinus block. The spinal fluid cell count was, 3,200 leukocytes, and cultures revealed growth of an anaerobic streptococcus. Sulfapyridine therapy was instituted. Coma ensued on December 23, and the patient died on December 29.

A postmortem examination of the brain and meninges was made. The leptomeninges were smooth and glistening. The spinal fluid was cloudy. Between the cerebellar hemispheres, on the inferior surface of the vermis, a thick tenacious whitish exudate covering an area of 2 cm. was discovered. No extradural or subdural abscesses or hemorrhages were found. The right lateral sinus was thrombosed to the level of the jugular foramen. The cranial nerves were examined with care. The only abnormality noted was enlargement of the right seventh and eighth cranial nerves so that they completely filled the internal auditory meatus.

Microscopic studies of the brain showed considerable small round cell infiltration of the subarachnoid space and proliferation of the arachnoid with areas of necrosis. The Virchow-Robin spaces were packed with small round cells. The right dorsal cochlear nucleus was composed of pale shrunken neurons with considerable pigment, eccentric nuclei and little or no Nissl substance—an acute tigrolytic reaction. The left facial nucleus and the left dorsal cochlear nucleus appeared normal. The pathologic diagnosis was subacute purulent meningitis and degenerative changes of the right seventh and eighth cranial nerve nuclei.

12. Leidler, R., and Sternberg, H.: Zur Klinik und Pathologie von Felsenbeintumoren, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **117**:186, 1928.

13. Nager, F. R.: Ueber die Knochenpathologie der Labyrinthkapsel, Acta oto-laryng. **26**:127, 1938

Microscopic examination of the serially sectioned right temporal bone showed it to be extensively destroyed by a neoplasm made up of groups of cells with an alveolar arrangement and separated into islands of various sizes by a connective tissue stroma.

The tympanic cavity, the mastoid area and the eustachian tube were filled by tumor (fig. 1). The tympanic membrane had been completely destroyed, and only a portion of the malleus was present. The bony walls of the eustachian tube were intact, and the canal of the tensor tympani muscle was not involved. The footplate of the stapes was dislocated at its posterior attachment and was displaced into the vestibule. A purulent exudate occupied the intracrural space of the stapes. Tumor tissue was observed adjacent to the anterior stapedial crus. The infralabyrinthine area, including the region of the bulb, was almost entirely

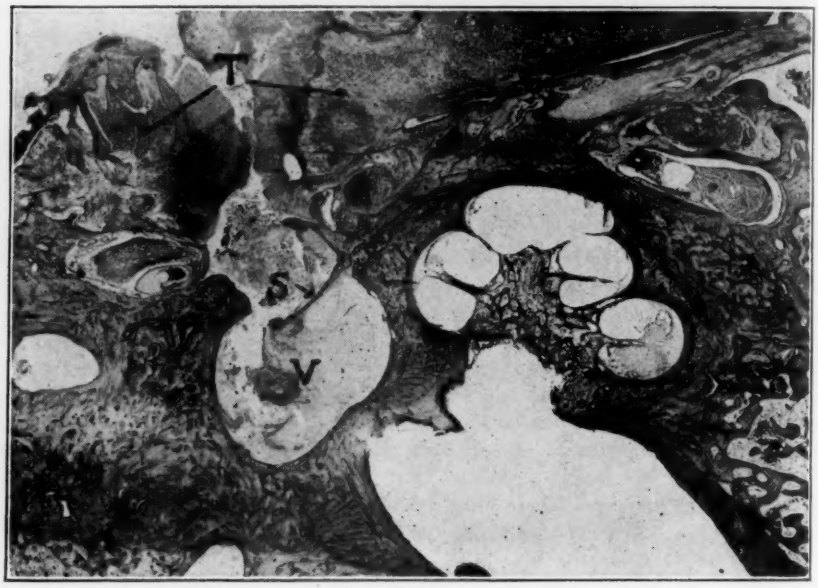

Fig. 1 (case 1).—Horizontal section (× 6.5) from the right ear of a patient with meningioma primary in the temporal bone. A large mass of the tumor (*T*) occupies the anterior portion of the middle ear and the eustachian tube. The footplate of the stapes (*S*) is dislocated from its posterior attachment. The vestibule (*V*) is nearly filled with a fibrinopurulent exudate. New bone formation is present in the basal coil of the cochlea.

destroyed by the tumor. The bony wall of the ascending carotid canal was eroded, and the carotid artery was compressed by the growth. At successively lower levels the tumor had extended widely into the soft tissues beneath the temporal bone. A small group of air cells extended posteromedially and posterosuperiorly from the tympanum into the apex, where there was evident both acute and chronic osteitis. Chronic osteomyelitis was present in the marrow of the tip. The membranous labyrinth was completely destroyed in the vestibule and showed beginning destruction, due to the suppurative process, in the cochlea. Purulent exudate was present in the osseous labyrinth and in the internal auditory meatus.

COMMENT

This is an example of non-reticulin-forming or non-collagen-forming meningothelial meningioma with the cells in alveolar arrangement. At times this type of tumor has been designated as alveolar sarcoma. Since the greatest portion of the tumor was found in the infralabyrinthine area, it may be assumed that the neoplasm originated from an arachnoid granulation in the region of the foramen of the jugular vein. Intratympanic extension of the tumor probably incited the development of the suppurative otitis media and mastoiditis of 1930. At the time of admission to the University of Chicago Clinics the patient had obliteration of the right jugular vein, destruction of the eleventh and twelfth cranial nerves at their exit through the skull, extension of the tumor in the carotid canal, degeneration of the seventh cranial nerve, invasion of the tympanic cavity and probably early labyrinthitis from neoplastic invasion to explain the absence of auditory function. The differentiation between chronic suppurative disease and tumor of the temporal bone could not be made until tissue was obtained at the operation. The source of the meningitis has not been definitely established, but the acute suppurative process present within the labyrinth suggests a labyrinthine origin. The histologic appearance suggests that the stapes may have been subluxated by packing used to control the hemorrhage and that thereby acute labyrinthitis was superimposed on a chronic process from tumor invasion.

The osseous labyrinth showed relatively little neoplastic destruction. It was apparently an important factor in limiting the growth to the tympanic and infralabyrinthine areas. The dural resistance prevented intracranial extension, and only on microscopic examination of the temporal bones was the final diagnosis made.

SARCOMA

The temporal bone is occasionally the site of origin of a malignant type of sarcoma. Several petrosal sarcomas were encountered in children and young adults. They are rapidly infiltrating, produce multiple cranial nerve palsies and usually result in death from intracranial extension. Occasionally a diagnosis may definitely be made by biopsy after the tumor has extended into the nasopharynx. Death usually ensues before metastases occur.

A sarcoma may be difficult to distinguish from a malignant type of fibroblastic meningioma (sarcomatous meningioma), the distinction being based on whether the tumor is encapsulated (meningioma) or infiltrative in character (sarcoma). In either case the prognosis is poor. Even though the encapsulated form is supposedly completely removed, it is apt to recur.[7] In Cushing's series of cases of sarcomatous meningioma the longest survival was two and one-half years.

CASE 2.—A malignant sarcoma primary in the petrosa with intracranial extension was observed in a 13 month old child admitted to the Bobs Roberts Hospital of the University of Chicago Clinics on Sept. 19, 1938 with right facial paralysis, irritability, anorexia and vomiting. Examination revealed papilledema of 1 diopter in the right eye, paralysis of the right external rectus muscle and ataxia in all movements of the head and the trunk. A diagnosis of pontile tumor was made, and roentgen therapy given. By September 22, the patient had become worse, and weakness, restlessness and vomiting had increased. By November 5 the voice had become hoarse, and by November 10 respirations had become labored. On November 13 myringotomy was done for acute otitis media of the right side. Roentgen examination on November 7 had shown extensive erosion of bone in the base of the skull about the apex of the right petrous pyramid. The patient died on November 14. The clinical diagnosis was sarcoma of the base of the skull.

Gross examination of the brain revealed on the inferior surface a large infiltrating mass, much more extensive on the right than on the left side, which had completely obscured the normal structure of the medulla, pons and hypothalamic region. Numerous nodules extended onto the inferior surface of the cerebellum along the margins of the vermis and the groove of the foramen magnum. Similar nodules extended along the sylvian fissures. On the left side small nodules were noted along the ninth, tenth and eleventh nerves. A section through the medulla and brain stem showed that the neoplasm extended 2 cm. into the cerebellomedullary angle. At this point the tumor had markedly deformed the medulla and had pushed the fourth ventricle well to the left.

The area of the right petrous pyramid was filled with a tumor mass measuring 5 by 3 by 2 cm., compared with a measurement of 3.5 by 2 by 2 cm. for the intact left petrous pyramid. The dura in both the middle and the posterior fossa was eroded.

Microscopic examination of sections stained with hematoxylin and eosin revealed in the tumor of the brain numerous large pale-staining cells with mitotic figures and numerous smaller basophilic cells. In the region of the pons the tumor had markedly compressed the brain tissue, and in some areas tumor cells were seen to invade the brain substance. In one area of the tumor, living bone with osteoclasts was observed. The tumor contained numerous strands of pink fibrous tissue. Sections prepared by Perdrau's method showed numerous dark-staining strands of connective tissue about the tumor cells. Fan-shaped areas of connective tissue invading normal brain substance were noted. The histologic diagnosis was fibrosarcoma primary in the petrous portion of the right temporal bone and extending through the dura to the medulla, brain stem, and cerebellum.

Histologic studies of the right temporal bone showed a large tumor mass occupying nearly the entire bone and distending the dura confining it. Microscopic examination of the tumor revealed it to be composed of bundles of tightly compressed and elongated cells which ran in various directions. Between the various bundles were large compact masses of cells rich in cytoplasm and with variably shaped nuclei. Mitotic figures were common. The tumor nearly filled the tympanic cavity (fig. 2). A small amount of fibrinous exudate was observed in the hypotympanum. The tympanic membrane was intact. The bony structure of the entire petrous pyramid was replaced by the tumor with the exception of the osseous labyrinth. The periosteal layer of the otic capsule was largely destroyed, but the enchondral and endosteal layers were almost completely intact. Beginning erosion of the promontory and of the medial wall of the epitympanum was observed. Active infiltration of the labyrinthine spaces had occurred.

DIFFUSE SARCOMATOSIS OF THE MENINGES

Sarcoma may arise in the meninges, spread through the cerebrospinal fluid and become implanted in multiple sites on the meninges. A case is presented of sarcomatous involvement of the eighth cranial nerve in the internal auditory meatus and multiple discrete sarcomatous lesions of the meninges. This tumor is rapidly growing and infiltrative in nature which distinguishes it from the rarely occurring meningiomatosis (Cushing)—the latter being characterized by multiple encapsulated

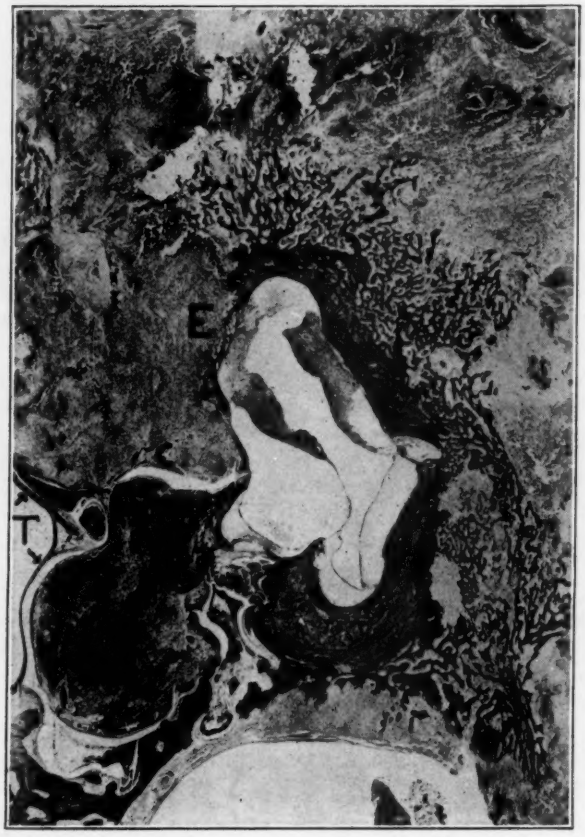

Fig. 2 (case 2).—Vertical section (× 5) from the right ear of a patient with primary sarcoma of the temporal bone. The middle ear is completely filled by the tumor. The tympanic membrane (*T*) is intact. Erosion of the medial wall of the epitympanum (*E*) is advanced, and tumor tissue occupies a portion of the vestibule, where it is limited in extent by the membranous labyrinth. Note the extensive erosion of the periosteal bone and the relative resistance of the enchondral and endosteal bone.

tumors of the acoustic nerves and meninges. When sarcoma involves the eighth nerve it may be confused for a time with neurofibroma (von Recklinghausen), acoustic neurinoma or even meningioma.

CASE 3.—L. J., aged 19, entered the University of Chicago Clinics on Oct. 29, 1931, complaining of severe occipital headaches and vomiting of two months' duration and diplopia of seven weeks' duration. Weakness of the left side of the face developed a week before admission.

Examination showed the left palpebral fissure widened, coarse nystagmus to the left, convergence absent, slight papilledema bilaterally with hemorrhagic optic disks, the left ophthalmic division of the fifth nerve paralyzed, muscle tone in the right upper extremity increased, abdominal reflexes more vigorous on the left than on the right side, and a Babinski sign present on the left side.

Suboccipital exploration, November 3, revealed a tumor lying in the fourth ventricle pushing the vermis of the cerebellum outward. The patient died on November 5 from respiratory failure.

Autopsy revealed the meninges in the cerebellopontile angles to be diffusely infiltrated with a gray tumor which extended forward to the optic chiasm. The leptomeninges of the spinal cord were infiltrated with tumor masses up to 1 cm. in diameter as far as the cauda equina. Median sagittal section of the brain showed that the tumor involved the meninges throughout the basilar cistern and extended up between the frontal lobes. It had completely surrounded the brain stem at the midbrain and extended over the corpora quadrigemina to the pineal body. Microscopically, the tumor was composed of spindle-shaped cells with scanty cytoplasm and oval nuclei. Mitoses were frequent. Silver impregnation did not show as much reticulin as is ordinarily found in a neurinoma. The phosphotungstic acid–hematoxylin stain showed no neuroglial fibrillae. There was no tendency to form collagen fibers. The pathologic diagnosis was diffuse sarcomatosis of the cerebral and spinal meninges. Sections of the temporal bones showed tumor masses infiltrating both auditory nerves but to a greater extent on the right (fig. 3). The tumor was made up of small spindle cells with small nuclei. Mitotic figures were occasionally observed. A striking feature was the presence bilaterally of a focus of otosclerosis in the region of the fissula ante fenestram; the foci were small, symmetric, evidently active and in an early stage. This observation recalls those which the late Dr. Albert Gray[14] recorded in 1933; he reported 3 cases of von Recklinghausen's disease with bilateral acoustic neurofibromatosis, and in 2 of the 3 cases he found new bone similar to otosclerosis in the labyrinthine capsule. Gray was under the impression that in these 2 cases the otosclerosis may have been "the result of pathological changes somewhere in the nervous mechanism which governs the nutrition of the structures of the cochlea, these changes being brought about by the extremely slowly destructive action of the growing tumor on the acoustic nerve, or, in other words, that it is a trophic change." He was of the opinion that otosclerosis is a trophic disturbance of the organ of hearing.

However, when the question is viewed in the light of observation made in other cases in which otosclerosis was diagnosed histologically and clinically at an early stage, it seems possible that either a local trophic or a prolonged systemic disturbance might provide the activating factor in the presence of a hereditary tendency.

GLIOMA

Neoplasms originating in the pons, e. g., glioma, may extend into the internal auditory meatus and interfere with the function of the seventh and eighth cranial nerves.

14. Gray, A.: Atlas of Otology, Glasgow, Jackson, Wylie Company, 1933, vol. 2; Two Cases of Acusticus Tumor with Clinical and Pathological Reports, Proc. Roy. Soc. Med. **26:** 1310, 1933.

CASE 4.—L. F., a girl aged 12, entered the University of Chicago Clinics Aug. 15, 1935 with a diagnosis of cerebellar tumor. Seven months previously her hearing and vision began to decrease. Two months later severe and persistent headaches developed. Three months before admission a staggering gait appeared, and a month later diplopia and vomiting became manifest.

On admission the following observations were made: right papilledema of 2.5 diopters, left papilledema of 3.0 diopters, bilateral paralysis of the abducens nerve, paralysis of the left facial nerve, absence of cochlear function on the left, absence of vestibular response to ice water on the left, sluggish pupils, sluggish speech, extreme dysdiadokokinesia, absence of superficial reflexes, and general hypotonia, more marked on the right.

Fig. 3 (case 3).—Horizontal section of the right temporal bone of a patient with diffuse sarcomatosis of the meninges. The internal auditory meatus is filled by the tumor, which is infiltrating the fibers of the eighth nerve (*T*). Particular attention is called to the otosclerotic focus (*O*).

On August 17 a suboccipital exposure of both cerebellar hemispheres was made. In the left cerebellopontile angle a soft tumor was found, which the neurosurgeon, Dr. Paul Bucy, believed was a lateral extension of a primary tumor of the pons. The patient died two days later.

At necropsy the pons was found to be considerably enlarged by an extensive tumor mass. The emerging cranial nerves were flattened. A small mass of tumor tissue extended out from the pons and followed the left acoustic nerve into the internal auditory meatus. Microscopic examination revealed the tumor to be made up of highly anaplastic cells varying in size and shape. Mitoses were frequent. A few multinucleated giant cells were present. Vascularity was pronounced. The tumor was not sharply demarcated but rather diffused into the normal brain tissue. The pathologic diagnosis was glioblastoma multiforme.

On histologic examination of the left temporal bone the internal auditory meatus was noted to be dilated by the tumor extending from the pons. The auditory nerve was compressed by the tumor, which extended to the cribriform plate. Alterations were present in the membranous structures of the cochlea, particularly Reissner's membrane, the tectorial membrane and Corti's organ, suggestive of recent antemortem degeneration (fig. 4).

VON RECKLINGHAUSEN'S DISEASE

Persons afflicted with this disease may have bilateral acoustic neurofibroma in association with the general involvement of other portions of the nervous system. The tumors of the patients are derived from connective tissue cell elements which develop between the fibers of the

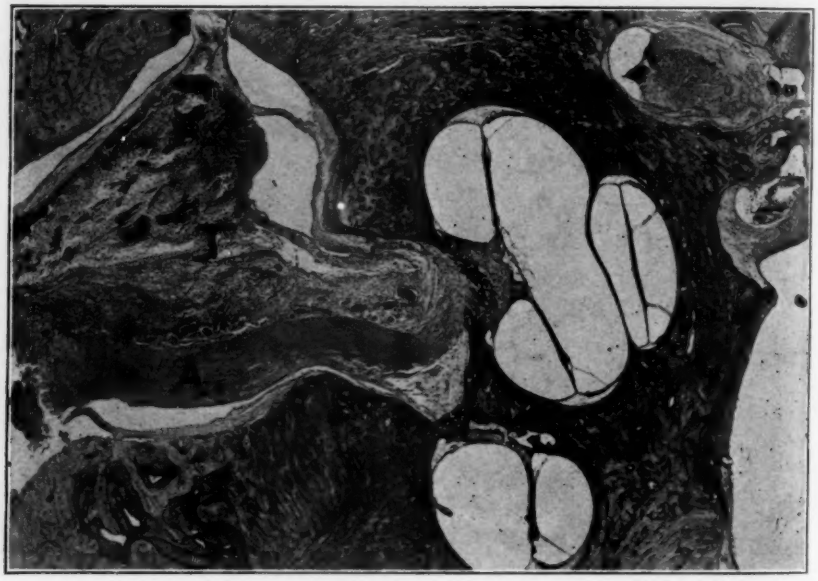

Fig. 4 (case 4).—Vertical section (× 9) of the left temporal bone of a patient who died of glioblastoma multiforme of the pons. This tumor extended into the internal auditory meatus. The meatus is dilated by the expanding tumor (*T*). The auditory nerve (*A*) is compressed. The membranous structures within the cochlea exhibit changes indicative of antemortem degeneration.

acoustic nerve without pushing them aside. They are distinguished from neurinomas by the fact that the latter grow in the auditory nerve and push the fibers aside. Acoustic neurinomas are relatively much more common, and their description will be reserved for a separate report. Meningioma of the internal meatus on the other hand arises from the meninges. Fowler [15] has called attention to the fact that certain acoustic tumors, especially meningioma, may originate from

15. Fowler, E. P., Jr.: Acoustic Tumors Within the Internal Auditory Meatus, Laryngoscope **46**:616, 1936.

the psammoma bodies and enlarged arachnoid villi commonly found within the internal auditory meatus. A case of bilateral neurofibromatosis is included in this report in which psammoma bodies were observed in both of the internal auditory meatuses. A similar observation was reported by Scott [9]—the unilateral presence of a large number of psammoma bodies in a case of bilateral neurofibroma. Since these psammoma bodies are usually found in meningiomas, their presence in conjunction with neurofibromatosis suggests a possible closer relationship between meningiomas and neurofibromatosis than has previously been suspected.

CASE 5.—J. H., aged 21, was admitted to the University of Chicago Clinics Dec. 16, 1937, complaining of weakness of the right arm and leg, ringing of the left ear and deafness of the left side, all of four years' duration. For the past six months he had weakness of the right side of the face, slurring of speech, attacks of blindness and a tendency to fall to the right.

Examination revealed atrophy of the right arm and shoulder, multiple subcutaneous nodules over the entire body, slurred speech, bilateral papilledema of 4.5 diopters, enlarged blindspots, absence of the left corneal reflex, diminished right corneal reflex, total loss of cochlear and vestibular function on the left (normal on the right), spontaneous nystagmus in the direction of gaze, spontaneous past pointing with the left hand to the right, tremor of the lips and the left eyebrow, husky voice, slight weakness of the right side of the face and positive Romberg sign. Roentgen studies showed calcification of the choroid plexus, slight enlargement of the left optic foramen and funnel-shaped enlargement of the left internal acoustic meatus.

Suboccipital craniotomy was performed on the left side by Dr. Paul Bucy on Dec. 30, 1937 and a large tumor removed from the left cerebellopontile angle. The patient died the following day. Necropsy, limited to the head, revealed the glial proliferations of von Recklinghausen's disease and multiple meningiomatous and neurinomatous involvement of the brain and cranial nerves.

Histopathologic studies of the left temporal bone showed neurofibromatous involvement of the cochlear and vestibular divisions of the eighth nerve, of the facial nerve and of the ninth and tenth cranial nerves. The internal auditory meatus and the facial canal were dilated. Operative intervention did not succeed in completely removing the tumor from the region of the cribriform plate. Large masses of psammoma bodies were observed in the internal meatus not only in juxtaposition to the meninges at the periphery of the nerves but also within the substance of the nerve trunks themselves.

Studies of the right temporal bone showed a similar neurofibromatous involvement of the cochlear and vestibular divisions of the eighth nerve, extending into the cochlea and into the fallopian canal as far as its descending mastoid portion. A small cluster of psammoma bodies was found adjacent to the cribriform plate.

METASTATIC TUMORS

The marrow spaces of the petrosa are occasionally the site of a neoplasm which has originated from a distant primary site. The marrow is capable of filtering out tumor cells from the circulating blood and giving rise to a metastatic growth. This process is similar to that by which miliary tuberculosis develops in the petrosal marrow in cases of miliary tuberculosis with an active primary focus in the lung.[6]

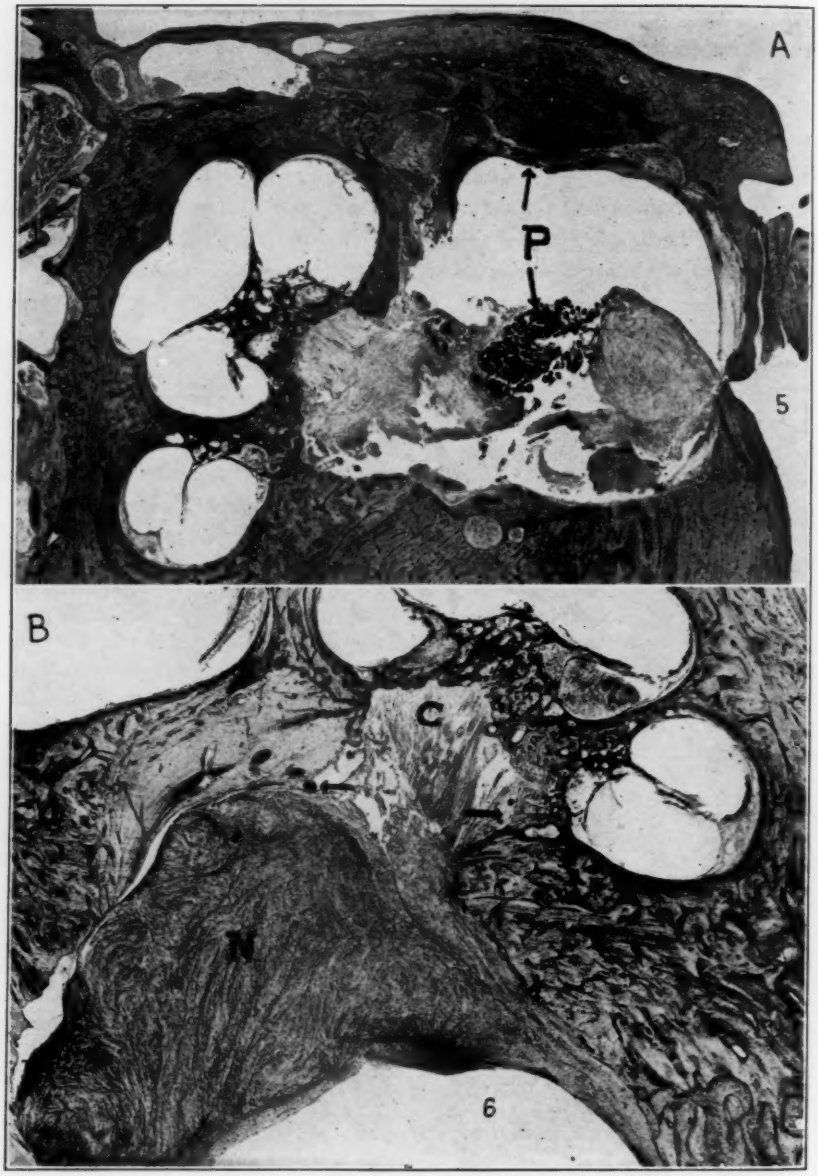

Fig. 5 (case 5).—*A*, vertical section (× 7) of the left temporal bone of a patient with bilateral neurofibromatosis (von Recklinghausen's disease), showing residual neurofibroma after a large neurofibroma had been surgically removed from the posterior fossa. Note the numerous psammoma bodies (pacchionian bodies) (*P*) in the dilated internal auditory meatus, not only along the superior margin of the meatus, where they are in intimate contact with the meninges, but also in the midportion of the meatus, where they are surrounded by neurofibromatous tissue.

B, horizontal section (× 10) of the right temporal bone (opposite from that in *A*). Marked dilatation of the internal auditory meatus has occurred. The meatus is occupied by typical neurofibroma (*N*). Calcified psammoma bodies are depicted in the fundus of the meatus (arrows). There is advanced atrophy of the remnants of the cochlear nerve (*C*) adjacent to the cribiform plate. The scala tympani in the second coil of the cochlea is nearly filled by the neurofibroma.

Metastatic tumors have rarely been observed in temporal bones not only because of the infrequency of histopathologic studies of temporal bones but also because of the fact that in the adult, the petrosal marrow, especially during that period of life when cancer is more likely to occur, has changed from red to fatty marrow. Case 6 (fig. 6) illustrates a metastatic focus of lymphosarcoma in the petrosal marrow of a patient who died with a primary lymphosarcoma of the cecum.

SUMMARY

The petrous portion of the temporal bone may be involved by tumors originating within the temporal bone, by tumors arising from adjacent structures and by tumors metastasizing from distant primary foci.

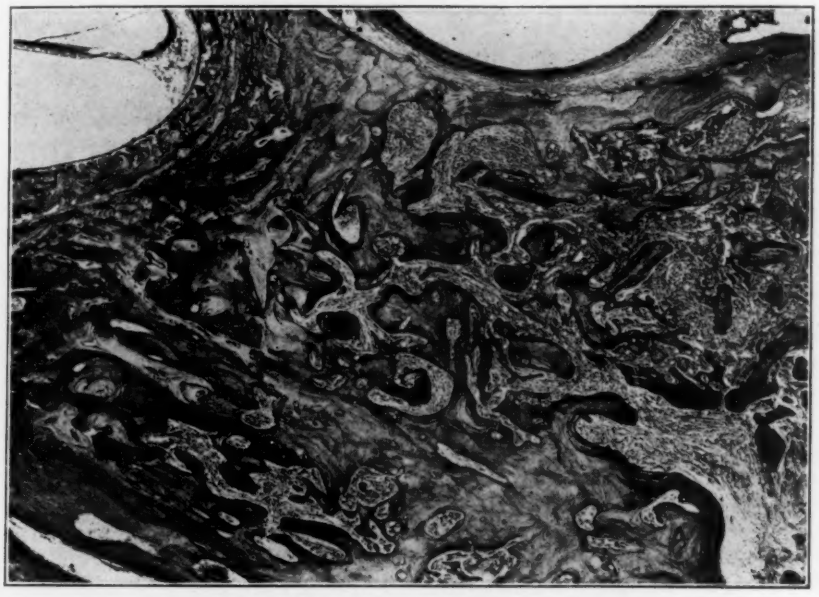

Fig. 6 (case 6).—Horizontal section (× 13) of the right temporal bone presenting a metastasis of lymphosarcoma of the cecum in the marrow spaces of the petrous apex.

The origin and the pathologic characteristics of meningioma involving the temporal bone are discussed. A case of meningioma of the temporal bone, complicated by acute labyrinthitis superimposed on chronic labyrinthitis produced by the tumor, is presented.

A case of sarcoma of the temporal bone is presented, and the pathologic characteristics of the tumor are pointed out. Occasionally a tumor of this type is confused with meningioma.

The salient features of diffuse sarcomatosis of the meninges are briefly discussed and illustrated. When sarcomatosis involves the

eighth nerve it may be clinically confused with neurofibroma, acoustic neurinoma or meningioma. In the case of diffuse sarcomatosis presented in this report, a small focus of otosclerosis was found in each labyrinthine capsule in addition to bilateral involvement of the eighth nerve.

Neoplasms originating in the pons, e. g. glioma, may extend into the internal auditory meatus and interfere with the functions of the seventh and eighth cranial nerves.

A case of bilateral neurofibromatosis of the eighth cranial nerve is presented in which psammoma bodies were observed in both of the internal auditory meatuses. Since these psammoma bodies are usually found associated with meningioma, their presence in conjunction with neurofibromatosis suggests a possible closer relationship between meningioma and neurofibromatosis than has previously been suspected.

One case of metastatic tumor of the temporal bone is presented. The bone marrow of the petrosa is capable of filtering out tumor cells circulating in the blood stream and becoming a secondary focus for a new growth.

1419 David Whitney Buliding (Dr. Proctor).

MALIGNANT MELANOMA OF THE NASAL MUCOSA

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ST. AUGUSTINE, FLA.

IN THE study of oncology, melanomas, the group of tumors characterized by melanin-pigmented cells, form a distinctive class of neoplasms that present vexing problems of remarkable interest, with wide differences of opinion regarding their origin and nature. These tumors may be innocuous or malignant. The benign tumor is commonly called a nevus or mole. The malignant tumor, frequently called a malignant melanoma and sometimes a melanotic sarcoma, is in practice generally designated simply as a melanoma, according to Boyd [1]; the old term "melanotic sarcoma" is a misnomer, for the tumor is not a sarcoma. Ewing [2] regarded the attempt to identify this tumor with carcinoma or sarcoma as undesirable. He advocated employment of the term "melanoma" to emphasize the specific character of the tumor cells and suggested that the possibility that a tumor of this type has its origin from epidermal cells be indicated by the term "melanocarcinoma" and a sarcomatous character by the term "melanosarcoma."

The nevus is usually but not always pigmented. It may occur on any part of the body but most frequently is situated on the face, the neck or the back. Rarely it is observed in the pigmented part of the eye. As a rule, its course is uneventful, but a mole exposed to long-continued friction or irritation is liable to become malignant. The malignant melanoma arises often in a nevus of the skin; in approximately one third of the cases it arises in the pigmented eye, where it is not preceded by a benign lesion. Nevi and melanomas occur proportionately in most regions of the body, but on the feet and genitalia, where moles are rarely present, melanomas commonly arise, apparently not from moles.[1]

Likewise, it would appear that melanoma of the nasal mucosa does not arise from a nevus, for the presence in the nasal fossa of this benign lesion has never been confirmed.[3] Consequently, the problem

1. Boyd, W.: A Text-Book of Pathology, ed. 4, Philadelphia, Lea & Febiger, 1943, pp. 42 and 291.

2. Ewing, J.: Neoplastic Diseases: A Treatise on Tumors, ed. 3, Philadelphia, W. B. Saunders Company, 1928.

3. Mayoux, R., and Perron, R.: Les tumeurs mélaniques du nez, Rev. de laryng. **60**:245-272 (March) 1939.

of its transformation into a malignant tumor is not a consideration here. Indeed, pigment is lacking in the mucosa of the whole lower portion of the nasal fossa; it is only at the level of the upper olfactory portion that the presence of pigment has been established.

This disease is truly rare and of particular importance because melanomas develop in definite sites where the tissues normally contain pigment.[4] The first case was reported by Viennois[5] in 1873. From a careful survey of the literature it appears that 66 cases have been recorded. Relatively few, however, have been described in British and American literature. In Great Britain, Wilkinson[6] reported the first case in 1912; 6 cases have since been published, 1 each by Mollison[7] (1920), de Santi[8] (1925), Collins[9] (1930), Tweedie[10] (1933), Jobson[11] (1934) and McKenzie[12] (1939). Smuts,[13] from South Africa, in 1939, reported a melanoma observed in a European at Capetown. In the United States, Coakley,[5b] reviewing the literature when he reported 1 case in 1907, mentioned 3 cases previously reported in this country, those of Lincoln[14] (1885), Shallcross[15] (1892) and

4. (*a*) Cuzenza, G.: Sui melanomi, Valsalva **8**:81-88 (Feb.) 1932. (*b*) Cozzolino, V.: Un caso di melanosarcoma della mucosa nasale: Studio clinico-istologico, Napoli, A. Trani, 1902; (*c*) Melanosarcome der Nasenschleimhaut mit Untersuchungen über die Entstehung des melanotischen Pigments: Klinisch-histologischer Beitrag, Arch. f. Laryng. u. Rhin. **15**:77-93, 1903. (*d*) Ewing.[2]

5. (*a*) Viennois, cited by Coakley. (*b*) Coakley, C. G.: Primary Melano-Sarcoma of the Nose: Report of a Case and Review of the Literature, Tr. Am. Laryng., Rhin. & Otol. Soc., 1907, pp. 430-441.

6. Wilkinson, G.: A Case of Melanotic Sarcoma of the Nose, J. Laryng. & Otol. **27**:1-8 (Jan.) 1912.

7. Mollison, W. M.: A Case of Melanotic Sarcoma of the Nose, Proc. Roy. Soc. Med. (Sect. Laryng.) **13**:39, 1919-1920; Internat. Zentralbl. f. Laryng., 1921, p. 174.

8. De Santi, P. R. W.: Melanotic Sarcoma of the Septum Nasi, Brit. M. J. **1**:109-110 (Jan. 17) 1925.

9. Collins, E. G.: A Case of Melanoma of the Nose, J. Laryng. & Otol. **45**: 691-693 (Oct.) 1930.

10. Tweedie, A. R.: A Case of Melanotic Sarcoma of the Nose, J. Laryng. & Otol. **48**:417-421 (June) 1933.

11. Jobson, G. B.: Melanotic Sarcoma of the Nose, Proc. Roy. Soc. Med. **27**:381, 1934.

12. McKenzie, W. S.: A Case of Melanoma of the Nose, J. Laryng. & Otol. **54**:93-94 (Feb.) 1939.

13. Smuts, P. A.: Melanotic Sarcoma of the Nasal Septum, South African M. J. **13**:287-288 (April 22) 1939.

14. Lincoln, R. P.: A Case of Melano-Sarcoma of the Nose Cured by Galvano-Cauterization, New York M. J. **42**:406-407 (Oct. 10) 1885; cited by Coakley.[5b]

15. Shallcross, I. G.: A Case of Melanotic Sarcoma of the Nares, Tr. Homeop. M. Soc. Pennsylvania (1891) **27**:212-215, 1892; Hahneman Monthly **27**:24-27 (Jan.) 1892; cited by Coakley.[5b]

Bristow [16] (1900). In recent years, Clevenger and Norris [17] reported 1 case in 1930, New and Havens [18] 1 case in 1932, Smith [19] 2 cases in 1939 and Kaplan [20] 2 cases in 1942. The case herein presented is apparently the eleventh reported in the United States and the nineteenth in the English language. Recently, four Japanese authors reported 1 case each, Cho [21] in 1937, Sato [22] and Ueki [23] in 1940 and Rin [24] in 1941 From Sweden, Ringertz [25] in 1938 reported 6 cases and summarized reports of 36 others which he had noted in the literature. In 1934 Penna de Azevedo and Guedes de Mélo [26] reported a case from Brazil, the tumor occurring in an Italian. The remaining cases were published by French, German and Italian writers, the largest number by Italian authors.

The incidence is approximately the same for the two sexes, and the disease is usually observed in persons of middle age. Mayoux and Perron [3] mentioned that, of 25 patients 14 were men and 11 were women. In discussing the case reported by Coakley,[5b] Richardson mentioned a melanoma he had observed in a child 4 years old, who died with involvement of the pharynx six months after operation, but the clinical diagnosis was not confirmed. In the literature reviewed, I noted that 34 of the tumors occurred in women and 29 in men; in the reports of 3 cases the sex was not mentioned. The ages available in 60 of the 66 cases averaged 58 years for the men and 59 years for the women, the extremes being 84 and 32 years for the men and 81 and 37 years for the women.

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16. Bristow, A. T.: Sarcoma of the Nasal Mucous Membrane, Brooklyn M. J. **14**:814-823, 1900; cited by Coakley.[5b]

17. Clevenger, W. F., and Norris, H. L.: Melanosarcoma Originating in the Mucous Membrane of the Lower Turbinate, Arch. Otolaryng. **12**:81-83 (July) 1930.

18. New, G. B., and Havens, F. L.: Fibroma, Carcinoma and Melanoma of the Nose and Nasopharynx, S. Clin. North America **12**:939-945 (Aug.) 1932.

19. Smith, A. T.: Primary Melanoma of the Nasal Cavity: Report of Two Cases, Arch. Otolaryng. **29**:437-445 (March) 1939.

20. Kaplan, I. I.: Malignant Melanoma of the Nose, Arch. Otolaryng. **35**: 85-90 (Jan.) 1942.

21. Cho, K.: Ein Fall von disseminierten Melanosarkomen in der Nasenhöhle, Oto-rhino-laryng. **10**:910 (Oct.) 1937.

22. Sato, I.: Ein geheilter Fall von Melanosarkom der Nase, Oto-rhino-laryng. (Abstr. Sect.) January 1940, vol. 13.

23. Ueki, I.: Re ein Fall von Melanosarkom der Nasenhöhle und Osteosarkom des Oberkieferbeins, Oto-rhino-laryng. (Abstr. Sect.) **13**:19-20 (April) 1940.

24. Rin, T.: Ein Fall von Melanosarkom der Nasenhöhle, Oto-rhino-laryng. (Abstr. Sect.) **14**:16 (April) 1941.

25. Ringertz, N.: Pathology of Malignant Tumors Arising in the Nasal and Paranasal Cavities and Maxilla, Acta oto-laryng., 1938, supp. 27, pp. 1-405.

26. Penna de Azevedo, A., and Guedes de Mélo: Melanoma primário do nariz, Arq. Inst. Penido Burnier **3**:48-56 (March) 1934.

Although common among the Sudanese, melanoma is particularly rare in American Negroes.[1] Kaplan [20] observed that of 40 tumors diagnosed as melanoma during a period of seventeen years at the New York City Cancer Institute, 4 were in Negroes, but none occurred in the nasal mucosa. This author and his associates had never encountered a malignant melanoma in a Negro patient on their service at Bellevue Hospital, where many New York Negroes are cared for, and Freed [20] informed him that in the literature of the preceding six decades but 31 cases of melanoma occurring in the Negro had been reported. An extensive review of the literature revealed that no case of melanoma of the nasal mucosa of a Negro had been reported up to the present time. In the case herein described the patient was a Negro woman, aged 81. It is of interest that the normal amount of melanin in the skin is much greater in the Negro than in the white man, although not exceeding 1 Gm. of pigment at most. Too, it is worthy of note that cancer of the skin is particularly rare among colored races.

ORIGIN

Through the years the question of the source of malignant melanoma occurring in the nose has been a matter of controversy. Wilkinson,[6] presenting the earliest report in the British literature in 1912, remarked that it was then generally accepted that these tumors arise in certain cells, stellate or spindle in form, occurring in the skin and the choroid and less frequently in the mucous membranes of the nose and the mouth. Named chromatophores, these cells were first described by Abdy [6] in 1889, he added, and their function is to produce pigment. They occur in the skin of Negroes and of animals with deeply pigmented skins and in the pigmented parts of the skin of white men, such as the areola of the nipple and in moles. Kaplan [20] noted that the olfactory apparatus consists of sensory cells containing granules that resemble the Merkel-Ranvier cells of the skin, which are associated with melanoma there, and that the supporting cells of the olfactory mucosa contain pigment accepted by Plaut as melanin.

In the opinion of Ewing [2] the melanoma is a pigmented tumor arising from a specific mesoblastic cell, the chromatophore, or from tactile cells lying in the epidermis or from nerve cells in the dermis. He regarded the Merkel-Ranvier cells as probably epithelial and the chromatophores as probably specialized cells belonging to a special embryologic physiologic order appropriated by the sensory nerve filaments as a peculiar end organ. This author concluded that theoretic considerations favor the view that all melanomas originate from the mesoblastic chromatophores and that human tumors originate from epithelial cells that have assumed pigmentary functions, adding that the cells have many specific properties which may resemble those of either epithelial or mesoblastic cells.

The early writers generally held the view that neurofibrous neoplasms, particularly neurosarcomas, arise from endoneural connnective tissue, being therefore mesodermal, but in 1910 Verocay [27] established that they are derived from proliferation of the cells of the sheath of Schwann and are almost certainly ectodermal. The classic studies of Masson,[28] published during the period from 1926 to 1932 and since amply confirmed, established conclusively that the nevus is of nervous origin, the cell being neuroectodermal and not epithelial in origin. The nerves of the skin terminate in two specialized end organs; the sheath of Schwann becomes the Meissner corpuscle in the dermis; the nonmedullated fibers, entering the basal layers of the epithelium, where they are known as Langhans' cells, spread out around the Merkel-Ranvier cells and the chromatophores, or melanoblasts. Although the characteristic groups of nevus cells bear no resemblance to the original neural cells, this investigator proved that they arise from a neoplastic proliferation of these specialized end organs and demonstrated the transitional stages. There is close association with both medullated and nonmedullated nerve fibers. Too, the neural cell is capable of differentiating to pigment formation, which is merely an expression of changed metabolic activity and in no way essential. Both in primary and in metastatic tumors, pigment may be completely absent or present in great concentration.[27] The variation in pigmentation depends on whether the nevus cell, nonpigmented and associated with the nerve endings, or the melanoblast, which has acquired the power of producing melanin, has a greater part in its formation.[1]

Histologically, it is also worthy of note that Theobald [27] in 1937 established that a nevus may be associated with the ciliary nerves both in the choroid and in the scleral canal. Duke-Elder [27] concluded that the deeper cells of the limbal conjunctiva and corresponding cells of the lacrimal caruncle are potential melanoblasts, which correspond to the chromogenic cells of Langhans of the skin. In discussing the two types of cells characterizing melanomas, the typical spindle-shaped cells of Schwann and the pigmented cells free of all Schwannian appearance, he observed that "it would appear that pigmented cells can represent a peculiar differentiation of neural elements, which is assumed by them in neoplastic conditions." He added that Theobald,[27] studying histologically 7 typical malignant tumors of the choroid, demonstrated their neural origin from the proliferating Schwann cells of the sheaths of the ciliary nerves traversing this tissue. He was of the opinion that nevi may be considered as an example of atavism, which explains their congenital and hereditary character.

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27. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1941, vol. 3, pp. 2461-66.

28. Boyd.[1] Duke-Elder.[27]

The wide divergence of opinion among pathologists regarding the origin of melanomas is illustrated by evidence submitted in favor of four different modes of origin summarized by Smith.[19] They are: (1) from the basal epithelium by a process of emigration and depigmentation (Dawson), with uveal melanomas explained on the ground that pigmented cells of the choroid are embryologically derived from the pigmented epithelium of the retina while melanomas of the adrenal and other deep organs are assumed to arise from pigmented neuroepithelium; (2) from fibroblasts by modification (Riecke and Pini), with some observers (Lowenbach, Walsch, Herxhermer, Bowman, Moller, Fox and Johnson) upholding the theory of endothelial origin of some melanomas while admitting that others are derived from epithelium; (3) from the end apparatus of cutaneous sensory nerves (Masson), and (4) from a highly specialized mesodermal cell, the melanoblast, or chromatophore (Ehrman and Ribbert), with the chromatophore regarded as a specific mesoblastic cell controlling production and transport of pigment, in terms of which this theory regards all manifestations of melanoma.

PATHOLOGIC ASPECT

The melanoma presents a picture so varied that it may exhibit carcinomatous or sarcomatous structure or both and may also simulate an endothelioma or even a lymphosarcoma (Boyd[1]; Ewing[2]). When malignant change takes place in a nevus, the cells of the tumor extend into the deeper tissues and become larger. The nuclei become hyperchromatic, and mitotic figures are present. The characteristic alveolar grouping of the large polyhedral cells in the fully formed melanoma, with the groups separated by a fine stroma, suggests a carcinoma. In the eye a sarcomatous arrangement with fusiform cells as in a fibrosarcoma is usually observed.[1] Collet, Mayoux, Perron and Girard[29] noted that in the nose the malignant tumor may have the aspect of a pseudosarcomatous tumor, an epithelioma or an endocrine tumor or of all three, with the pseudosarcomatous appearance present most frequently.

Pigmentation, usually pronounced, may be absent altogether, or absent in one portion of the tumor and present in excessive degree in another. When a melanoma is without pigment, the dopa reaction determines the presence of enzyme in cells which have not yet formed melanin. Phagocytic histiocytes take up the pigment liberated by the pigment-bearing cells that break down. The tumor appears black to the naked eye, but the intracellular pigment is yellow.

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29. Collet, F. J.; Mayoux, R.; Perron, and Girard: Étude histologique des tumeurs mélaniques du nez, Ann. d'oto-laryng., September 1938, pp. 797-811.

Usually the melanoma remains small, but the widespread metastases it produces make it particularly dangerous. It spreads at first by the lymphatics to the regional lymph nodes, but late in the course of the disease the tumor cells may be widely disseminated throughout the body by the blood stream as the fatal termination approaches. Metastases occur commonly in the skin. Ringertz [25] noted only 2 cases recorded in the literature in which melanoma metastasized to the nasal cavity; both were clear cases of primary melanoma of the skin with multiple metastases.

NASAL SITE

Mayoux and Perron [3] stated that in cases of melanotic tumor of the upper part of the digestive tract and the respiratory passages, localization on the nasal mucosa occurs fairly frequently; of 38 cases mentioned, the site in 42.7 per cent was the nose and sinuses. The nasal mucosa has a pigment-free and a generously pigmented area. In the upper portion of the nasal cavity the epithelium of the olfactory mucosa is filled with an abundant supply of granular pigment. It would seem reasonable that this upper nasal mucosa with its wealth of pigment should offer a site as favorable for the development of a melanoma as the richly pigmented surfaces of other parts of the body, such as the pigmented nevi of the skin and the uveal tract of the eye,[30] and certainly one more favorable than the lower pigment-free portion. This is, however, not the case, for these tumors arise in the mucosa that is without pigment, either in the septum or in the lateral wall, including the turbinate. Pipia [31] stated that malignant melanoma never arises in the superior portion of the nasal membrane; it emanates only from the septum and the two lower turbinates, but not from the upper turbinate. In 63 of the cases mentioned in the literature, the initial site was given as the nasal cavity in 17, the septum in 21, the inferior turbinate in 14, the middle turbinate in 4, the middle and inferior turbinates in 1, the lateral wall in 4, the floor of the nose in 1 and the external ala in 1. In the case presented the melanoma arose in the inferior turbinate.

Wilkinson [6] stated that in his case the site of origin was probably close to the junction of the skin of the vestibule with the mucous membrane of the outer wall of the nose, where infiltration of the underlying tissues is greatest. Since the skin of the vestibule is often pigmented, he concluded that there appears to be no reason why melanomas should not occur as often there as elsewhere on the skin. Collins [9] also regarded the probable origin as in the mucocutaneous junction of the vestibule of the nose. Smith [19] noted that malignant melanoma is to

30. Jacob, L.: Das Melanosarkom der Nase, Inaug. Dissert., Leipzig, B. Georgi, 1900.

31. Pipia, I.: Un nuovo caso di sarcoma melanotico del naso a punto di impianto sul setto, Valsalva **1**:424-432, 1925; Ann. di laring. **3**:161-168, 1927.

be considered in cases in which papilloma occurs in the anterior portion of the nasal cavity, springing either from the turbinates or from the floor of the nose. He concluded that the mucous membrane in this region probably contains misplaced tissue or cell rests of cutaneous origin, which may give rise to a tumor of this type, and mentioned similarity to the not infrequent origin of melanoma in the rectum, another area of transition from cutaneous to mucosal surface. Briani [32] was inclined to the theory of origin from melanoblasts of the skin of the vestibule fortuitously included in the nasal mucosa in aberrant form.

Cozzolino,[4c] writing in 1903, was of the opinion that when a malignant melanoma is present on the high portion of the nasal septum, the source of the pigment is easily found in the mucosa of the olfactory region, which, according to Todd and Bowman,[33] contains a yellowish granular pigment in the deep layers of its epithelium. Cuzenza [4a] considered that the tumor may develop from aberrant pigmented germs, according to the Durante-Cohenheim theory. In his investigations he was unable to observe that pigmentation was present in the nasal mucosa, even in the olfactory zone. He concluded, therefore, that pigmentation of the nasal mucosa is not a constant finding and that the occurrence of melanoma at this site loses its importance if one admits that the nasal mucosa contains pigment.

In their histologic studies of multiple specimens, Mayoux and Perron [3] were able to recognize adjoining sarcomatous and epitheliomatous aspects, thus confirming in the case of nasal melanomas the work of Masson [28] and others on the cutaneous melanotic tumors. Penna de Azevedo and Guedes de Mélo [26] also made observations in the histologic study of their case of primary melanoma of the nasal fossa in accord with the conception of Masson,[28] finding the blastomatous elements oriented or distributed along the trunks or branches of the nerves.

CAUSE

Mayoux and Perron [3] observed that melanotic tumors of the nose are always malignant, since there is no nevus of the nasal mucosa, and that they seem to require no special etiologic conditions. They mentioned that the one peculiar feature of these nasal tumors is the importance of the added inflammatory phenomena. Etiologically, the most interesting question to them was whether or not melanomas graft themselves on other lesions. Replying to several observers who stated that the melanotic tumor develops following ablation of ordinary polyps, they concluded that the reverse is true, the polyps arising from the tumor at the moment when the inflammatory characteristics overbalance, and then

32. Briani, A.: Melanoma primitivo del naso, Gior. med. d'Alto Adige **7**:496-506, (Oct.) 1935.

33. Todd, R. B., and Bowman, W.: The Physiological Anatomy and Physiology of Man, London, J. W. Parker, 1856, vol. 2, p. 37.

hiding the true tumor, as in Michael's cases.[34] They added that the pathogenesis remains wholly unexplained, as does the curious fact that melanotic pigment does not appear in the nose except in these malignant growths.

Pipia,[31] discussing the uncertain etiology of these tumors, mentioned the apparent influence of traumatism in his and other cases[35] and suggested that the trauma of operation may transform benign lesions into malignant tumors in patients subjected to repeated removal of polyps. In reporting his case, Bilancioni[35] stressed the coincidence of a gunshot wound involving the malar region and the eye of the same side and a melanotic tumor of the nasal cavity appearing several months later. He was of the opinion that since the ocular bulb was pierced, it was not only possible but probable that choroid chromatophores were carried from the eye to the nasal mucosa In the case reported by Margarot, Rimbaud and Ravoire[36] the first lesion, on the external ala of the nose, was associated with a lead shot carried there for several years.

SYMPTOMS

Usually the first symptom is unilateral nasal blockage, which is soon associated with recurrent epistaxis. Occasionally, the hemorrhage is frequent and profuse. Pain is not often a symptom unless the tumor has grown to the extent that there is pressure, or unless there is blockage of a sinus. The discharge of a blackish or inky-black serous fluid is frequently present. Only occasionally in the literature is there mention of such symptoms as light headache, orbitofrontal headache, a sense of heaviness in the head and neuralgia of the frontal sinuses or orbits. Since the lesion usually remains small, its importance lies not in any local symptoms it may produce but rather in the widespread metastasis which ensues. It may, however, grow until nasal deformity is present, and a blackish mass may protrude from the naris.

In 53 of the cases recorded in the literature, symptoms were noted as follows: for one week, several weeks, one month, seven months, twenty months, three years, four years, five years, ten years and fourteen or fifteen years in 1 case each; for a short period, several months, two months, six months, eight months, ten months and eighteen months in 2 cases each; for four months in 4 cases; for one year in 4 cases; for three months in 8 cases, and for two years in 10 cases. In 1 case

34. Michael: Melanosarkome der Nase, Verhandl. d. internat. med. Cong. (pt. 12) **4**:83, 1890; cited by Coakley.[5b]

35. Bilancioni, G.: Sarcoma melanotico della fossa nasale sinistra sorto alcun tempo dopo ferita da fucile che aveva interessato la regione malare e l'occhio dello stresso lato, Arch. ital. di laring. **41**:5-26, 1921.

36. Margarot, J.; Rimbaud, P., and Ravoire, J.: Mélanome malin développé autour d' un corps étranger et suivi d' une généralisation immédiate, Bull. Assoc. franç. p. l'étude du cancer **28**:448-455 (March) 1939.

nasal obstruction was described as ancient, and in 2 cases the symptom of bleeding was described as recurrent. In 48 cases, the symptoms mentioned were bleeding in 8 cases, bleeding and obstruction in 16 cases, obstruction in 19 cases, swelling in 3 cases, swelling and obstruction in 1 case and swelling, obstruction and bleeding in 1 case. Thus obstruction was a symptom in 37, or 77 per cent, bleeding in 25, or 52 per cent, and swelling in 4, or 8 per cent, of the cases in which this information was available. In 18, or 37.5 per cent, of these cases there was a history of removal of polyps.

DIAGNOSIS

The melanoma is variously described as a mushy purple, reddish gray, reddish black or black growth, a cauliflower mass bleeding readily on the slightest manipulation and a tumor resembling a ripe mulberry. Kaplan [20] commented that the mere presence of a pigmented area in the mucous membrane offers no conclusive evidence for a diagnosis of this tumor, since pigmentation is common in the Negro and other dark-skinned peoples. Histologic study is essential.

Although pigmentation is usually present in great degree, it is not an invariable criterion in the diagnosis of this lesion. The histologic picture without the assistance of melanin may have to be the guide. The dopa reaction is of value in these instances for it indicates those cells which are true melanoblasts, containing the oxidase ferment capable of transforming the colorless chromogenic material in the blood into melanin.[1] In the first case described by Smith,[19] pigmentation was present in the primary lesion but not in the metastatic node in the submaxillary region; in the second case, pigment was not present in the primary tumor on the inferior turbinate, but there was definite pigmentation in the metastatic submaxillary node.

Ringertz [25] was of the opinion that it is uncertain whether or not there are malignant nasal and paranasal melanomas which are melanin-free from the outset. He noted, however, that recurring tumors of this type may contain practically no pigment. This author concluded that the pigment present in the tumor must be analyzed before the diagnosis of malignant melanoma can be considered established and pointed out that Heymann,[37] as early as 1888, and many later authors directed attention to the possibility of hemosiderin-pigmented tumors being erroneously identified as malignant melanomas, especially since melanomas are often very vascular and contain many hemorrhages. The readiness with which these growths simulate other malignant tumors has already been mentioned.

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37. Heymann, P.: Melanotisches Sarkom der Nase mit Demostration, Tagebl. d. Versamml. deutsch. Naturf. u. Aerzte **61**:248-249, 1888; Ueber Melanosarkome der Nase, Internat. Zentralbl. f. Laryng. **8**:310, 1891.

Denker [38] stated that in cases of malignant tumor of the nose with metastases, which develop by way of the lymphatics, the diagnostician must also look for enlargement of glands in the retropharyngeal and submaxillary regions and lying in the depths on the jugular vein. Zahler [39] described a case illustrating this point even though the original tumor and the metastases occurred on opposite sides. Kedrowa,[40] too, reported a case in which the formation of metastases passed from one side of the body to the other.

TREATMENT

Radiotherapy seems to be generally unavailing. Irradiation was employed in addition to surgical intervention in 18, or 43 per cent, of the 42 cases reviewed in which therapeutic measures were described in any detail, without appreciable benefit. Metastasis or recurrence appears to take place regardless of treatment.

The slow growth of the primary tumor as compared with the rapid dissemination of the metastatic lesions is an important consideration in treatment. Simple removal of the tumor, although apparently complete and followed by irradiation with radium or roentgen rays, seems to be of no permanent benefit. Radical removal including a wide margin of normal tissue would appear, however, to offer a reasonable chance of cure. Often in a nasal operation, such removal cannot be performed without resulting deformity and impairment of function, but it should nevertheless be undertaken even though the lesion is small.[19]

Once the diagnosis has been confirmed by careful histologic study, wide exposure and removal should be carried out promptly by an external approach. Two methods of approach have proved feasible, as noted by Smith,[19] lateral rhinotomy, or the Moure operation, with the incision being made through the cheek along the side of the nose, or the Rouge, or Denker, operation, with the incision beneath the upper lip. In the latter procedure he found ligation of the external carotid artery sufficiently helpful to warrant the extra trouble, although many surgeons regard it as unnecessary. Ueki,[23] in performing the Moure operation, also resorted to ligation of this artery.

PROGNOSIS

The prognosis is grave, but the outlook is perhaps not so serious as usually stated, in the opinion of Boyd.[1] In some cases the tumor remains localized over a long period. This author stated that even after involvement of the lymph nodes has taken place, cure may follow removal of

38. Denker, A.: Ueber Behandlung der bösartigen Nasengeschwülste, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **122**:45 (Aug.) 1929.

39. Zahler, A.: Ein Melanosarkom der Nase, Monatschr. f. Ohrenh. **66**:1416-1418, 1932.

40. Kedrowa: Melanosarcoma nasi, Zentralbl. f. Hals-, Nasen. u. Ohrenh. **13**: 568, 1929.

the primary tumor together with the enlarged glands and the intervening lymphatics.

The average duration of the disease is two to three years, according to Collins [9]; Tweedie [10] estimated it at four years after the first symptom appears. Heymann [37] reported recurrence of the tumor in his case after four years. A number of the cases surveyed in the literature were either not traced or were followed only for a relatively short period. A clear record for one year following treatment was noted in less than one sixth of all the cases reviewed. Aside from Heymann's case, in only 2 cases, those of Sato [22] and Smuts,[13] was there a record of a patient's surviving without recurrence or metastasis for more than two years.

Observing that the duration of this disease is brief and that it is well known to be fatal, Clevenger and Norris [17] remarked that death usually ensues from metastatic involvement of the liver or other parts of the body. In the case published by Wolffheim,[41] in which the melanoma emanated from the septum and recurred on the floor of the nose and in the lumbar part of the spinal column after repeated operations, autopsy showed universal formation of melanoma. Autopsy ten months postoperatively in the case of Lannois and Jacod [42] showed extensive metastases in the liver and the spinal cord. In 1 case reported by Ringertz [25] metastases were noted in the lungs and the abdominal organs at autopsy six weeks postoperatively, and in another generalized distant metastases were observed at autopsy one and one-half years after treatment had begun.

In Smuts's [13] case the primary lesion was in the septum, with involvement of the cervical glands of the left side and later involvement of the glands of the right side. Ineffectual radium therapy was followed by excision of the growth and mass dissection of the glands of the left side, with block dissection of the glands of the right side five months later. For approximately two years thereafter the patient was in perfect health. At autopsy, following his death from acute lobar pneumonia and acute pericarditis, there was no evidence of melanoma. Kaplan [20] estimated that metastasis occurs in about 50 per cent of the cases.

REPORT OF A CASE

C. W., a Negro woman aged 81, was examined on Jan. 15, 1938. She complained of blockage of the left nasal passage, which had been present for the three months immediately preceding examination, with recurrent bleeding since she had "picked a sore" in the left nostril two months before. The bleeding had become increasingly profuse, she related, until oozing was almost constant.

41. Wolffheim, W.: Melanosarcomatos universal von einem Septum-sarkom ausegehend, Ztschr. f. Laryng., Rhin. **11**:1-3, 1922.

42. Lannois and Jacod: Sur un cas de sarcome mélanique du nez, Ann. d. mal. de l'oreille, du larynx **41**:694-702, 1922; Rev. de laryng. **44**:355-362, 1923.

When the nose was examined, the left nostril was filled with a dark, reddish black, smooth growth, the surface of which bled readily on probing. It was not attached to the septum or to the floor of the nose. A probe was passed around it without difficulty. It took its origin in pedunculated form from the junction of the anterior and middle thirds of the left inferior turbinate. The growth had not extended into the nasopharynx. The general appearance of the tumor suggested

Fig. 1.—This photomicrograph of a portion of the tumor shows the modified squamous epithelial lining of the turbinate. The submucous stroma is filled with tumor cells and pigment. The intense cellularity and the nuclear variations are evident. Involvement of the subepithelial chromatophore apparatus is indicated. The epithelial pearl formation is suggestive, but not well brought out here. × 10.

that it had been treated with the popular Negro remedy of spider webs and soot. The right side of the nasal cavity was entirely normal.

On general physical examination no glandular enlargement was noted, nor was there other evidence of metastasis. The blood pressure was 190 systolic and

110 diastolic. The total and differential blood counts were within normal limits, and the Wassermann test of the blood was negative. Urinalysis revealed the presence of fine granular and hyaline casts, a trace of albumin and a few pus cells. Roentgen studies of the chest and sinuses yielded no evidence of pathologic change.

With a snare a section of the tumor was removed for biopsy. The report of the pathologist (L. Y. Dyrenforth) who examined the specimen follows:

Fig. 2.—The periacinic arrangement of the tumor cells and the large amount of brownish yellow pigment, both intracellular and extracellular, are shown in this photomicrograph of a portion of the tumor. × 10.

"Gross Description: The specimen consists of several small irregular pieces of tissue of dark red color and uniform and friable consistency. Two of the smaller pieces have a grayish mottling on the outer surface.

"Microscopic Description: The appearance of the stained sections at once denotes a malignant growth. Likewise, the distribution and character of the

pigment, which is present in quantities, are such as to confirm a diagnosis of melanoma.

"Flat sheets of large, irregular cells are seen. Some are large and pale, others small and dark. Their outlines are roughly polygonal. Derivation from the chromatophore elements of the dermis is indicated by the tumor cell involvement of this layer. Fairly rapid proliferation is evident from the poor differentiation

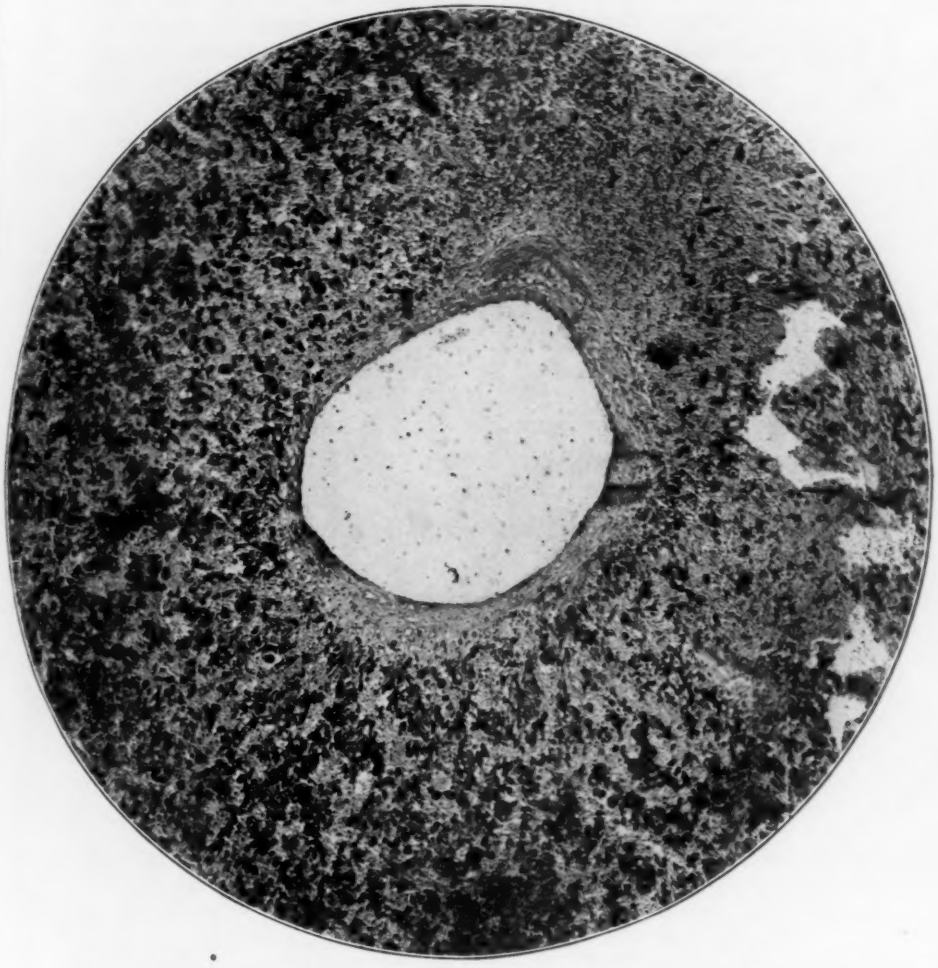

Fig. 3.—The periacinic configuration is evident in this photomicrograph. Stromal hemorrhage is diffused throughout the lower segment. × 10.

displayed. A faint periacinar or perivascular arrangement is suggested. [See figure 1.]

"The epithelial origin is more prominently displayed than is usual with malignant melanomas, because of a persistent tendency to form epithelial pearls.

"The pigment is present as small rounded globules of yellowish brown, finely granulated material. It is unevenly distributed in rather ample cytoplasm. [See figures 2 and 3.] Mitotic figures are not frequent. Most of the nuclei are large,

pale and vesicular, with prominent nucleoli. No antecedent tissue structure is recognizable.

"Diagnosis: malignant melanoma; melanocarcinoma."

The patient was kept under observation in the hospital for three days and then discharged, with the bleeding stopped. She failed to return until four months later when, on May 18, she again reported to the hospital, complaining of epistaxis, now more profuse and frequent than when she was first examined. There was a constant dripping of a brownish red discharge. No change in the size of the tumor was apparent. The patient was kept under observation in the hospital for two days at this time, and then she disappeared. Efforts to trace her were unavailing.

SUMMARY

Malignant melanoma of the nasal mucosa occurs rarely and is unique in that it develops in a site where the tissues normally do not contain pigment.

Views of various authors regarding the origin, the nature, the site and the pathologic and etiologic aspects of this disease are presented. The symptoms, the diagnosis, the treatment and the prognosis are described.

The case reported apparently is the first one of a melanoma occurring in a Negro to be recorded in the literature. A survey revealed reports of 66 cases, only 10 of which were published in this country. These cases are analyzed with respect to sex, age, duration and nature of symptoms, site within the nasal cavity, treatment and prognosis.

EUSTACHIAN TUBE

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MONTREAL, CANADA

AURAL disease, particularly disease of the middle ear, is so prevalent that an intimate knowledge of the pathway of infection is necessary to the otologist.

The causes of otitis media, although numerous, may be summarized in the one phrase: "Obstructed and infected noses."[1] As a general rule, contrary to the theory accepted by the laity, the tympanic membrane and the middle ear do not become infected through the external canal. In at least 95 per cent of cases infection spreads to the middle ear and the tympanic membrane by way of the eustachian tube. The importance of this tube is at once apparent, as it is the only portal of entry. Although its structure and relationship are clearly described in present day textbooks, one is apt to forget that little has been added to the knowledge of the eustachian tube during the past four hundred years. If any excuse is necessary for referring to the early anatomists, one should remember the words of Adam Politzer, who, in the preface to his history of aural therapy, said: "He who would claim complete knowledge in this respect, must learn the work of earlier times."

From the time of Herophilus until the sixteenth century careful anatomic investigation was almost at a standstill. Then followed a period of renewed activity due to the brilliance of the Italian school of anatomists. During this time Vesalius (1514-1564), Eustacrius (or Eustachi) (1524-1574) and Fallopius (or Fallopio) (1523-1562) paid particular attention to the anatomy of the ear, followed a hundred years later by Valsalva, Morgagni and Scarpa. Quoting from Imperatori,[2] Portal in his *Histoire de l'Anatomie* stated:

> Eustache has made important discoveries about the ear. He is the first who knew the canal, which on one side opens into the tympanum and on the other side into the back part of the nostrils. He has discovered the malleus muscle (tensor tympani) and indicated the origin and course of the nerve which winds in the interior of the drum (chorda tympani). He also has given an exact description of the limacon (snail) cochlea. The canal of communication between

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Presented before the Interne's Society of the Royal Victoria Hospital, June 15, 1946.

1. Laurens, G.: Oto-Rhino-Laryngology for the Student and Practitioner, Bristol, J. Wright & Sons, 1922, p. 60.

2. Imperatori, C. J.: Bartholomaeus Eustachius, Laryngoscope **53**:631-652 (Oct.) 1943.

the nose and the ear, he has said, has the form or shape of a quill. . . . Although Eustache could have claimed this discovery, as none of the ancient anatomists had spoken directly of this canal, he did not feel embarrassed to give it his name. Eustache has acquired an immortal reputation for himself and nobody has ever refuted that he did not discover the tube.

From the accurate description of the tube that bears the name of Eustachius and the engravings drawn over four centuries ago, one might easily imagine them taken from a modern textbook of otology. Although his account of the tube is the original,[3] there is a description written in 1616 by Helkiah Crooke,[4] taken from Eustachius, which perhaps is more explicit. Writing on the eustachian tube, Crooke said:

. . . Of this passage Aristotle made mention but Eustachius was the first that described it, and after Volcherus Coiter. This hole or passage is like a round pipe or small quill, larger in the beginning and passeth obliquely to the inward and foreside of the Basis of the braine, and in the middest of foure holes it penetrateth the whole bone where it is encreased (encased) by a substance that is partly gristly and partly membranous. This canale thus encreased with substance of another kind passeth between two muscles of the throate and neare the root of those processes which we likened to Bats wings, on the inside, I say, of them it determineth into one of the cavities of the nostrils and is inserted into the thicke coate of the Palat neare the root of the Uvala. But the inward extremity or end of this passage where it respecteth the middle cavity of the nostrils becometh a strong gristle bunching or swelling much outward, which is covered with the mucous or slimy coate of the nose, and set as a Porter to keep the end or outlet of the passage.

The figure of it is not round but a little depressed making 2 angles, the hollowness thereof is much about the proportion of a goose quill, yet so that it is twice as broad in the end as it is in the beginning, and is likewise covered with a mucous coate, but that thin. The termination of this passage is on both sides common to the nosethrilles. . . .

Furthermore, that the aire doth passe out of the mouth into the eares, we are taught by Aristotle in the eleventh Chapter of the first Booke of his History of Creatures, and also by experience, because when a man yawneth wide he cannot heare what is spoken, and beside he perceiveth a noyse in the eares. In like manner, when a man blowes his nose or with any force containes his breath, he shall ever perceive that the aire entereth into his eares, and with a certain perturbation whereby the implanted ayre is disturbed, because it is a drift not without some violence or constraint but when ayre insinuateth itselfe of its owne accord for the refection of that which is bredde in the eare, then is the motion gentle so that wee doe not perceive it. And beside the length of the passage prepareth and fitteth the newe ayre for the refection and nourishment of the old . . .

He that would find this passage must take a dryed skull and put a hogs bristle into the hole of hearing, he shall perceive that it will issue again in the palat of the mouth. But in a greene head the holes of this canale are very conspicuous in the same palat.

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3. Eustachius, B.: Opuscula anatomica, Lugd. Bat., J. vander Linden, 1707, pp. 138-140.

4. Crooke, H.: Microkosmographia: A Description of the Body of Man, London, M. Sparke, 1616, p. 586.

On reading through the description one cannot help but marvel at the anatomic and physiologic observations made by Eustachius with the limited means at his disposal. He noted that the tube is lined with mucous membrane throughout and is continuous with that of the nose. Mention is also made of the cartilaginous cushion at the pharyngeal orifice, covered with mucous membrane, which provides a natural protection of the tube and middle ear. Eustachius also described the method of opening the tubes usually credited to Valsalva. Concluding his remarks on the tube, he said:

> . . . the knowledge of this canal will be of extreme value to doctors in their right use of medicines, because in future they will know that even the most dense material can be discharged from the ears, not by narrow ducts, but by a broad passage either by an effort on the part of nature or by the help of medicines which are called masticatoria.

Over one hundred years later, Valsalva [5] suggested inflating the eustachian tube when he advised "his very best patients to hold their nose and blow out the cheeks" in order to clear their ears.

In 1863 Politzer [6] described his method of inflating by means of the bag which bears his name and the auscultation tube and said it was more effective than the eustachian catheter. In recent years further knowledge has been added.

Shortly after the last war the gross anatomy and physiology of the eustachian tube were described by Rich.[7] He summarized his findings as follows:

(1) normally the tubes are closed;

(2) the tensor veli palatini, innervated by the Vth nerve, is the only functional muscle of the tube;

(3) the tubes are opened during the swallowing, yawning and sneezing reflexes, and are best ventilated by the yawning reflex.

The importance of maintaining equalized air pressure on both sides of the drum was appreciated by the air force personnel during the first world war. Recent articles produced in the second world war indicate that aviation otologists stress the importance of the normal functioning of the eustachian tubes. Endurance of the rapid changes of altitude to which air crews are subjected, especially on descending,

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5. Valsalva, A. M.: De aure humana tractatus, Traj. ad Rhenum, G. vande Water, 1717.

6. Politzer, A.: Ueber ein neues Heilverfahren gegen Schwerhörigkeit in Folge von Unwegsamkeit der Eustachischen Ohrtrompete, Wien. med. Wchnschr. **13**:84, 102, 117 and 148, 1863.

7. Rich, A. R.: A Physiological Study of the Eustachian Tube and Its Related Muscles, Bull. Johns Hopkins Hosp. **31**:206-214 (June) 1920; The Innervation of the Tensor Veli Palatini and Levator Veli Palatini Muscles, ibid. **31**: 305-310 (Sept.) 1920.

is dependent on patent tubes which equalize the changing atmospheric pressure on the drums.

During the last few years there have appeared in the literature a number of papers on the eustachian tube. The microscopic anatomy and caliber of the tube have been carefully studied by Wolff.[8] Graves and Edwards[9] have more recently given a most interesting review of this topic.

A number of years ago I became interested in this subject while studying under Professor Nager in Zurich, Switzerland. Corrosion

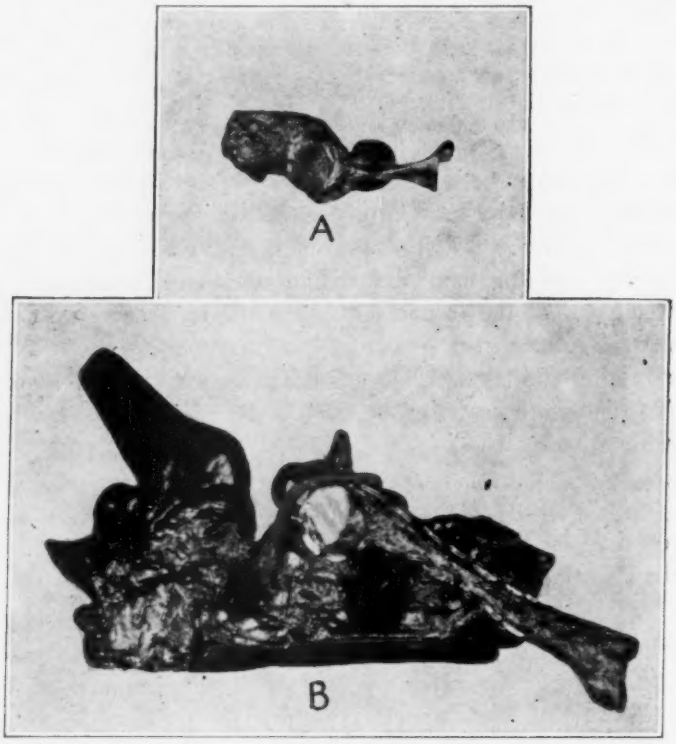

Fig. 1.—Corrosion casts of the temporal bone with the round middle ear and the eustachian tube dotted in. *A* shows the short, horizontal tube of the newborn infant and *B* the adult tube, just twice the length of, and at an angle of 45 degrees to, that of the infant. Both specimens are actual size. (From the author's collection in the Anatomical Museum of McGill University, Montreal, Canada.)

8. Wolff, D.: Microscopic Observations of the Eustachian Tube, Ann. Otol., Rhin. & Laryng. **40**:1055-1067 (Dec.) 1931; Microscopic Anatomy of the Eustachian Tube, ibid. **43**:483-494 (June) 1934; The Auditory or Eustachian Tube, Laryngoscope **53**:400-408 (June) 1943.

9. Graves, G. O., and Edwards, L. F.: The Eustachian Tube, Arch. Otolaryng. **39**:359-397 (May) 1944.

casts of the temporal bone were made in Wood's metal as described by Siebenmann,[10] who had been in charge of the clinic before Nager. Metal casts of dried bones showing the semicircular canals and the cochlea in relation to the fallopian canal and the cells of the mastoid process are not difficult to make but it requires great care to obtain the eustachian tube intact. By thoroughly drying the bone, especially the mastoid portion, and hardening the tube in some fixing agent, such as solution of formaldehyde U. S. P. or graded alcohol, satisfactory casts can be made. After the molten metal has beeen poured, the specimen is cooled and then immersed in weak hydrochloric acid. Unless the softened tissue is teased away gently and gradually, after the bone and the cartilage have dissolved, the tube is liable to break at the isthmus. The details of this method have been described elsewhere.[11]

A number of casts, as illustrated, made from soft tissue specimens, showed the eustachian tube at different ages. The sources of the specimens varied from newborn infants to aged persons.

The tube of the adult, as seen in figure 1*B*, is shaped like a trumpet at each end and is about 36 mm. in length. The narrow point, the isthmus, at the junction of the osseous and cartilaginous portions, is situated about 24 mm. from the pharyngeal opening of the tube. This constriction is fairly constant in size, the opening being about 4 mm. high by 2 mm. in width. The tube, at an angle of about 45 degrees, has the widened pharyngeal opening about 16 mm. below the tympanic orifice.

In newborn infants and young children, the tube is much shorter and more horizontal, and the lumen at the isthmus is not as large or well defined as in the adult. Some interesting measurements bearing on the length and the position of the tube in the child have been given by Symington.[12] Although not a recent publication, his article is worth quoting, as anatomy remains the same. He stated:

> The eustachian tube of the new-born child differs from that of the adult in its length, in the size of its lumen, in its direction, and in the condition of its walls. [He then recorded the accompanying table of measurements.]

Age of Subjects	Length of Tube
Two 9 mos. foetuses	17 mm. and 18 mm.
Child 6 weeks	17 mm.
Child 1 year	20 mm.
Child 1 year 3 months	20 mm.
Child 2 years	23 mm.
Child 4½ years	31 mm.
Child 5 years	30 mm.
Child 7 years	30 mm.
Three adults	35 mm., 35 mm. and 36 mm.

10. Siebenmann, F.: Korrosionanatomie, Jena, Gustav Fischer, 1897.

11. Tremble, G. E.: Two Anatomic Preparations of the Temporal Bone for Teaching Purposes, Arch. Otolaryng. **4**:412 (Nov.) 1926.

12. Symington, J.: The Topographical Anatomy of the Child, Edinburgh, E. & S. Livingstone, 1887, p. 50.

From this [the table of measurements] it appears that the tube doubles its length between infancy and adult life—an increase which is greater than that which occurs in the external auditory meatus. . . .

The tympanic orifice is certainly as large, if not larger in the child, than in the adult, but the pharyngeal opening is unquestionably much smaller. . . . The growth of the Eustachian tube seems to occur mainly in its anterior or pharyngeal portion. Its increase is associated with a projection of the posterior lip of the pharyngeal orifice on the lateral wall of the pharynx. In the new-born child, the tube is nearly horizontal and certainly does not form an angle of more than 10° with the horizon. In the child, aged four and a half years, the direction of the Eustachian tubes is much nearer that of the infant than of the adult, as the angle they form with the horizon is less than 20°, while in the adult the tubes incline so much downwards, as they pass from the tympanum to the pharynx, that they form an angle of at least 45° with the horizon. This descent

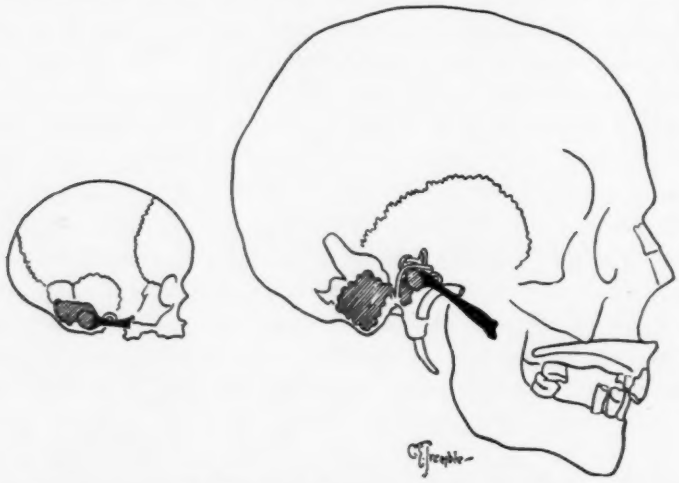

Fig. 2.—Diagrammatic drawing of corrosion casts showing the relative positions of the eustachian tube in the infant and the adult. For clearness the middle ear and the mastoid cells are shaded and the eustachian tube is blacked in.

of the tube does not keep pace with that of the floor of the nose; for, according to Kunkel,[13] the pharyngeal orifice of the tube lies below the level of the hard palate in the foetus; in the new-born child it is at the same level; at the age of four years 3 or 4 mm. above it, and in the adult 10 mm. above it. Von Tröltsch[13] described the adult Eustachian tube as being, on an average, 35 mm. in length, of which 24 mm. were cartilaginous and 11 mm. osseous. He contrasted the relative lengths of the osseous and cartilaginous portions in the Eustachian tube and external auditory meatus. While only one-third is cartilaginous and two-thirds osseous in the meatus; in the tube the reverse is the case. . . . In the new-born child there is scarcely any osseous Eustachian tube, and the outer wall of the tympanic orifice is bounded by the tympanic ring.

[In the adult] the outer part of the tube, which normally is open, has a rigid and comparatively non-elastic wall, while the pharyngeal end is collapsible. The

13. Cited by Hasse, C.: Anatomische Studien, Leipzig, W. Engelmann, 1869, vol. 1, no. 1.

mucous membrane of the closed tube lies in longitudinal folds, especially toward the pharynx and floor of the tube. Here the thickness of the submucous layer is much increased. When the tube is dilated, these rugae mostly disappear. The long diameter of the closed pharyngeal ostium measures, on the average, 7 mm. The open ostium, an equilateral triangle, averages 6 mm. in height and 6 mm. in width.[14]

Clinically, acute otitis media at the present time is not considered the serious infection it was even ten years ago. This is due in great measure to the widespread use of chemotherapy. It is now well recognized that the common cold is not influenced by sulfonamide compounds but that the complications occurring in the middle ear and the mastoid process by way of the tube are greatly diminished by their use. As a result, any one complaining of discomfort of the ear following acute coryza, sinusitis or infectious disease is immediately given chemotherapy. This tends to abort the infection in the early stages when it is due to gram-positive organisms so that in relatively few cases does it progress to abscess formation. The infrequency now of paracentesis and mastoidectomy corroborates the rationale of this form of treatment.

In cases of acute or chronic suppurative otitis media, the removal of the secretion is important. Some prefer to cleanse by simply mopping out the discharge with dry absorbent, while others recommend irrigation with sterile saline solution. When the secretion is scanty, the dry treatment is often sufficient, but if there is a profuse discharge, irrigation is usually less irritating.

The method employed is unimportant as long as the discharge is carefully and thoroughly removed from the external canal. In cases of acute otitis media, after the ear had been discharging from three to five days Politzer recommended inflating the eustachian tube to evacuate the pus more freely. He was convinced this improved the hearing, gave the patient a subjective feeling of relief and hastened the healing. In the presence of nasopharyngitis he used an antiseptic spray in the postnasal space before inflating the tube. When this conservative form of treatment is carried out, followed by instillation of mildly antiseptic drops, the secretion ceases in a few days and the perforation closes in the majority of cases. However, at times the discharge does not subside and then catheterization often helps. Furthermore, many chronically diseased ears are improved by this method in cases in which the character of the discharge suggests an infection of the eustachian tube. It is a well known fact that tubal infection as a result of otitis media is not dangerous from the point of view of mastoiditis but is troublesome and persistent.

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14. Bryant, W.: The Eustachian Tube: Its Anatomy and Its Movements, M. Rec. **71**:931-934 (June 8) 1907.

I have used a no. 2 silver catheter with a small funnel soldered to the upper surface and found it useful (fig. 3).

Two catheters are necessary, one for each side. A recent article by Weiss [15] suggested that an ordinary catheter may be used, with the patient's head tilted backward after the method of Politzer. When the catheter shown in figure 3 is used, the head is simply held in a comfortable upright position. After acute otitis has persisted for two to three weeks without symptoms of involvement of the mastoid process and the discharge has become thick and stringy, suggesting a low grade infection of the tube, the catheter described is helpful. At first iodine powder and various medicated drops, including a solution of tyrothricin, were tried in an attempt to clear up the infection of the tube. More recently, penicillin and streptomycin have been found more satisfactory.

Good results have been obtained by instilling a solution of a penicillin salt (5,000 units to 1 cc.) into the funnel after ascertaining that the tip of the catheter was in the pharyngeal opening of the tube. Before attaching the compressed air nozzle to the catheter, it is necessary to

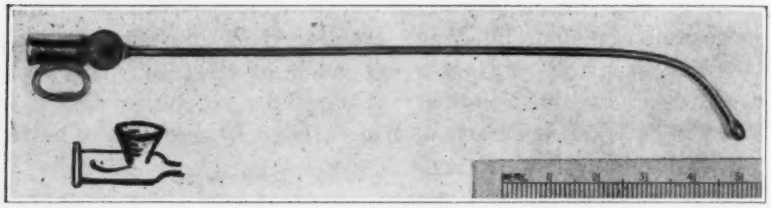


Fig. 3.—Silver catheter with a small funnel soldered to the upper surface, useful in the treatment of disease of the eustachian tube and middle ear.

close the top of the funnel with the left index finger, so that the solution will enter the orifice of the tube.

To obtain the best results, it is important to blow out any thick mucus or inspissated pus present in the tube before using penicillin. Unfortunately, although penicillin has given spectacular results in the armed services during the last three or four years and recently in the civilian population, its usefulness is limited to gram-positive organisms. It is true that the vast majority of infections of the middle ear are caused by strains of streptococci, staphylococci or pneumococci, but invariably cultures show a mixture of germs including gram-positive bacteria. The newer mold, streptomycin, is effective against gram-negative organisms, so that a combination of the two has been used with excellent results. Streptomycin is stable, retaining its effectiveness for months while penicillin loses its potency after a few weeks. Also,

15. Weiss, J. A.: Transtubal Instillation of Penicillin in Acute Otitis Media, Arch. Otolaryng. **43**:17 (Jan.) 1946.

streptomycin, like penicillin, can be given intramuscularly or used locally, and on a mucous membrane it causes little or no discomfort. Furthermore, they do not lose their individual properties when mixed together.

METHOD OF PROCEDURE

After politzerizing the affected ear to clear the eustachian tube, the external canal is mopped out repeatedly until it is perfectly dry. The head is then turned on the side, with the ear to be treated toward the ceiling, and the canal is filled with a solution of penicillin and streptomycin, equal parts. The patient is instructed to open the mouth widely and close it as in yawning, and occasionally to swallow in order to open the tube. At the same time the operator applies intermittent pressure to the tragus to close the canal, exerting pressure and slight suction which allows the solution to seep through the perforation into the middle ear. After pressure has been applied ten or twelve times a little absorbent material is placed in the concha, the catheter is inserted through the nostril after cocainization and the opening of the eustachian tube is found. A few drops of the solution of penicillin and streptomycin are instilled into the funnel by means of a dropper, and the nozzle of the compressed air pump is attached to the catheter after closing the funnel with the finger. The solution is blown through the catheter into the opening of the tube. This is repeated three or four times.

There might be some doubt as to whether the solution enters the tube to any appreciable extent. On the other hand, every otologist is consulted by patients suffering from otitis media who can hold the nose and blow mucus and pus through the tube and middle ear into the external canal. The discharge usually makes an audible sound as it emerges through the perforation. Also, patients with suppurative otitis media often remark that they taste drops prescribed by their physician when these are instilled into the ear. A little dye in the medication has shown that in many cases the solution can be worked through the tube by way of the external canal and also blown through from the inside by means of the catheter.

With this procedure being repeated every second day or sometimes just twice a week, many suppurative infected middle ears have dried up entirely that had resisted all other types of conservative treatment. In conjunction with this form of office treatment, patients are given drops of the same solution to instil into the affected ear three times daily after thoroughly drying the external canal.

When Valsalva remarked over two centuries ago that the eustachian tube "was of great help to doctors," he little realized the full import of his statement. However, since the days of the early anatomists, when gross dissections were the only means of investigating the human body, scientific progress has advanced considerably. The microscope, unknown to the ancients, has brought to light the minute anatomy, and the newer branches of bacteriology and biochemistry have broadened one's vision. In recent years, chemotherapy in the form of sulfonamide drugs has given spectacular results. With the discovery of penicillin and streptomycin, numerous other molds are being investigated, and this field shows great promise for the future. Fleming, during his visit, stressed the importance of the local use, as well as the systemic administration, of penicillin. This was also noted by his co-workers,

the Floreys.[16] Quoting from their article: " . . . the problem of using penicillin locally is that of devising some means to apply a very soluble and diffusible substance so that a bacteriostatic concentration is constantly maintained . . ." This is easily accomplished by means of the described catheter in cases of subacute and chronic otitis media in which the infection is limited to the tube. Spectacular results have been obtained by this method over the past two or three years in cases in which the infected ear did not respond to the normal methods of treatment. Considering the great advances in medicine of the past few decades, the modern otolaryngologist must feel indeed fortunate that so many of the infections in his sphere of activity can be diagnosed and controlled by chemotherapy.

1390 Sherbrooke Street, West.

16. Florey, M. E., and Florey, H. W.: General and Local Administration of Penicillin, Lancet **1**:387-397 (March 27) 1943.

EXPERIMENTAL INSOMNIA AND AUDITORY ACUITY

VICTOR GOODHILL, M.D.
AND
DAVID B. TYLER, Ph.D.
LOS ANGELES

IN JULY 1945, an investigation of the effect of prolonged wakefulness on the physiologic and psychologic efficiency of 12 healthy young men was conducted by one of us (D. B. T.). It was decided to include a study of auditory acuity among the various physiologic and psychologic tests performed. The subjects were volunteers from a Southern California Conscientious Objectors' Camp. They remained awake for one hundred and twelve hours, during which time they were kept busy with various types of physical and mental activities involving a considerable amount of heavy physical labor.

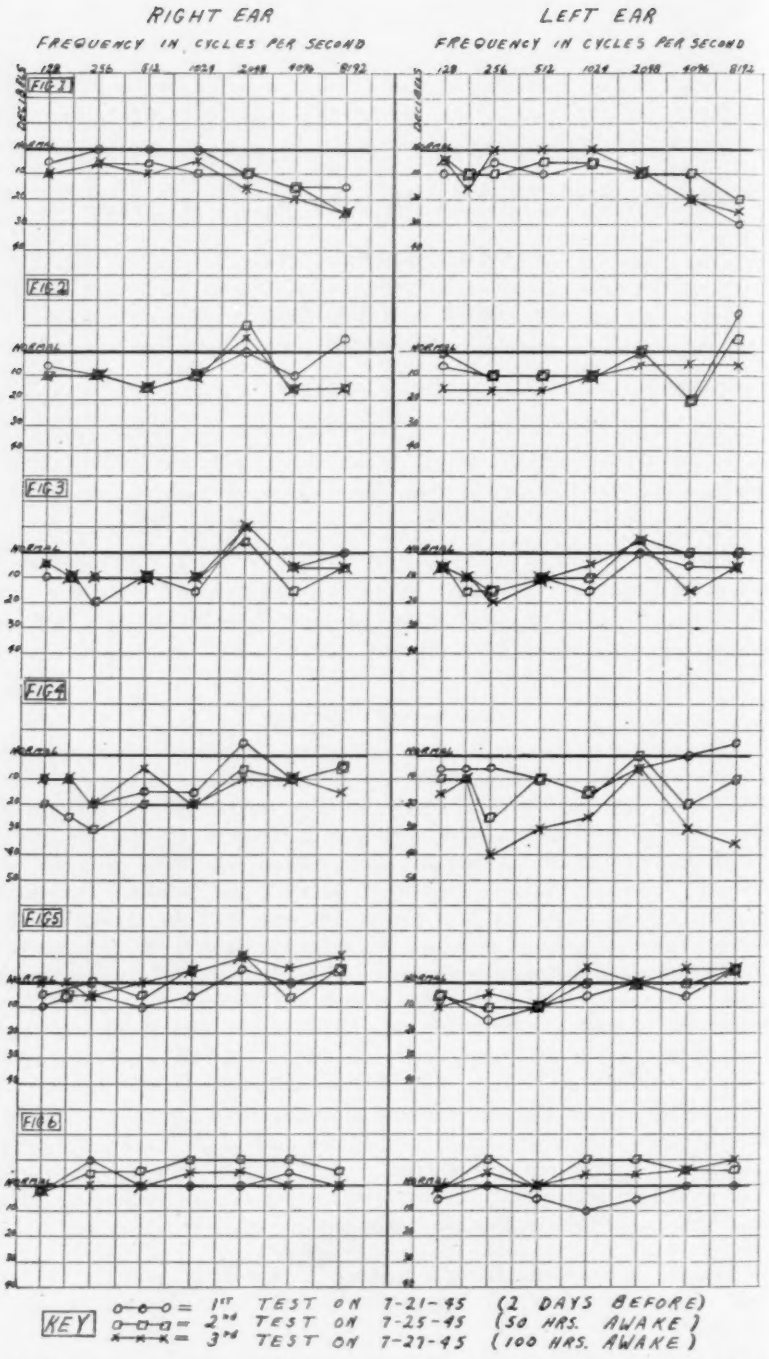
METHOD OF TESTING

It was decided to limit the testing method to audiometry. Ordinary speech tests with whispered and spoken voice are not quantitative measurements. Fork tests also are only qualitative in character. For the purposes of this test two standard clinical audiometers were employed, a Western Electric 2A (battery operated) and the Maico audiometer. Each subject was tested with the same machine by the same examiner in each of the three test periods. The subjects were all examined before the first test to determine that the external auditory canals were free of cerumen or debris. In all of the subjects the tympanic membranes were intact, although some presented evidence of old healed tympanic fibrosis. No active otitic disease was encountered in these men. (One of the subjects, no. 12, had a cold during the course of the experiment, and during the second test, at the end of fifty hours, he showed a definite conductive type of hearing impairment; the hearing returned to normal at one hundred hours.)

The testing method was the standard procedure advocated by the late C. C. Bunch. Threshold determinations were made for each of the 7 octaves of C commonly used in clinical otologic practice. Air conduction tests only were made. It had previously been determined that the hearing of each subject was within normal limits and that the Rinne test was positive on each side in each subject.

From the Department of Otolaryngology, University of Southern California School of Medicine, and the Department of Biology, California Institute of Technology.

The work described in this report was done under a contract recommended by the Committee on Medical Research, between the Office of Scientific Research and Development of the National Research Council and the California Institute of Technology.

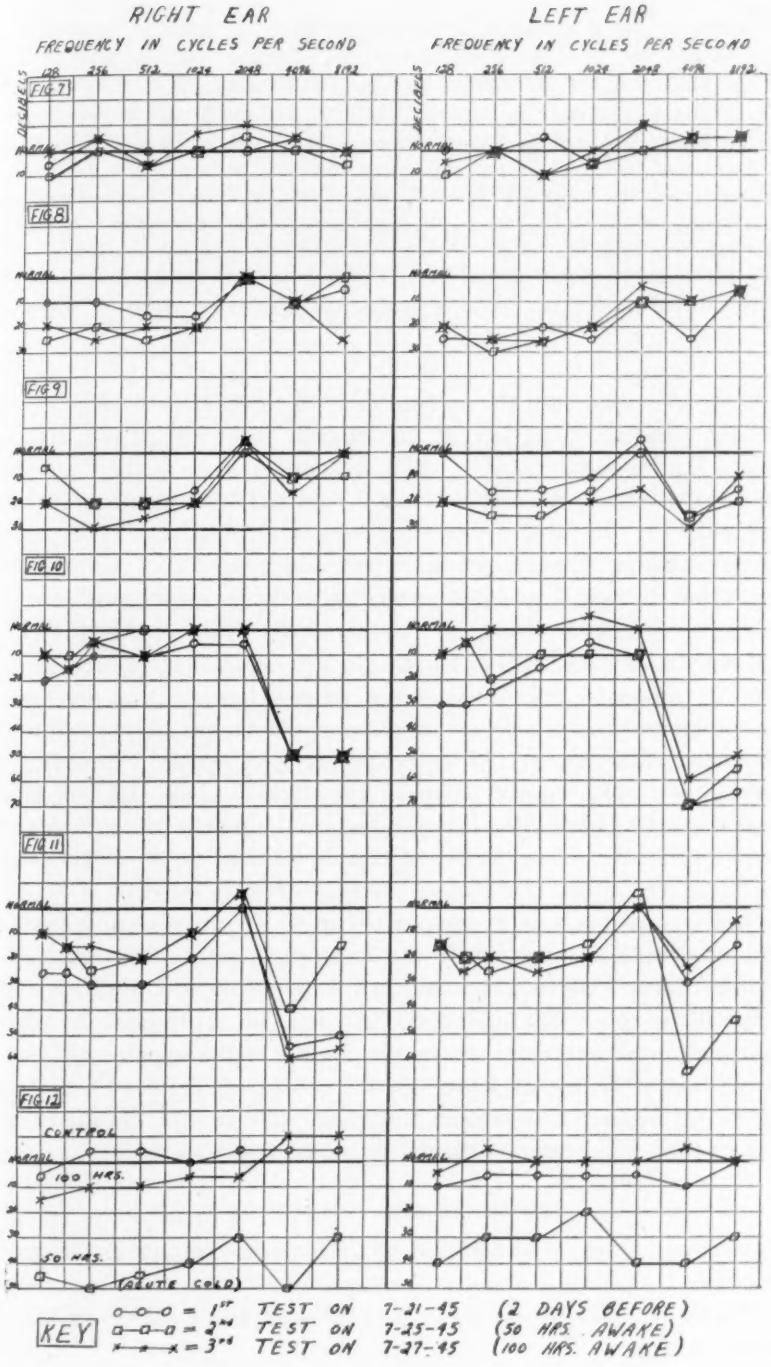


Audiograms of twelve subjects of experimental insomnia. Each audiogram shows three threshold curves made as follows:

July 21, 1945 (two days before onset of the experiment—a control), identified by circles.

July 25 (after subject had been awake for fifty hours), identified by squares.

July 27 (after subject had been awake for one hundred hours), identified by crosses.



Audiograms of the twelve subjects continued.

The technic employed consisted in the determination of threshold by averaging both crescendo and decrescendo intensities; i. e., ascending and descending intensities were both used, and the average threshold obtained was then recorded.

Observations were made as follows: The first, on July 21, was a control taken two days before the onset of the experiment; the second, on July 25, was taken two days after the onset of the experiment, or after approximately fifty hours of wakefulness; the third, on July 27, was taken on the one hundredth hour, approximately twelve hours before the conclusion of the experiment. In audiometric practice, variations of plus or minus 5 to 10 decibels are considered to be within the limits of error in the average patient. Accordingly, this physiologic variation was taken into consideration in the final analysis of results.

RESULTS

As can be seen from the audiograms, no significant hearing loss or gain was determined in any of the subjects after either fifty or one hundred hours of wakefulness. This was just as true of the 9 subjects with normal hearing as it was of the 3 who had high tone losses (nos. 9, 10 and 11). In these 3 men the losses were presumed to be due to previous cochlear damage caused by acoustic trauma. It is interesting to note that in these 3 subjects, as well as in the 9 subjects with normal hearing, no significant change in auditory acuity could be detected.

It was rather surprising to find this lack of decrease in auditory acuity following prolonged wakefulness. For years it has been the practice of some otologists to warn their patients to avoid undue fatigue before an audiometric examination (to insure proper psychologic cooperation in a subjective test); however, as seen here lack of sleep and inordinate fatigue appear to have little, if any, effect on the threshold determination of auditory acuity. As a matter of fact, in some of these subjects there is an apparent slight increase of acuity. This increase is of no real significance inasmuch as it may be either (1) a physiologic variation, which was mentioned previously or (2) an increase of acuity due to the learning process, which is encountered occasionally in clinical practice.

It will be noted that subject 12, in whom an acute infection of the respiratory tract developed during the course of the experiment, had a hearing loss at the fiftieth hour. At the end of one hundred hours, and in spite of the excessive fatigue and recent infection of the upper respiratory tract, his hearing returned to the normal level.

SUMMARY AND CONCLUSION

Auditory acuity was followed in 12 subjects during experimental insomnia.

Prolonged wakefulness up to one hundred hours had no effect on hearing acuity.

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MAGNETIC REMOVAL OF FOREIGN BODIES FROM THE FOOD AND AIR PASSAGES

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THE GENERAL Electric Company a few years ago introduced a new magnet. Ths magnet is an alloy of aluminum, nickel, cobalt and iron, fused under high pressure at almost the melting point, in reducing gas. Of the several such alloys, the most powerful synthesized is "cast Alnico–V." which can lift twenty-five times its own weight. Because the alloy is brittle, it is most useful if the fusion is done in a mold of the desired size and shape. Should the magnet ever lose its virtue, it can be remagnetized.

Although magnets were used more than a century ago in attempts to remove metallic objects from the human body, success was not often achieved until recent years. Electromagnets have proved successful in removing metallic splinters and other fragments from the eye, but in general magnets have been clumsy, cumbersome and expensive. The Alnico magnet, however, can be made small enough to be passed through even a 4 mm. bronchoscope. Although the Alnico magnet was offered for ophthalmologic use in 1943, the first report of its being used to remove foreign bodies from the food and air passages appeared a year later.

My first model of the magnet, a cylinder 3.5 cm. long and 0.5 cm. in diameter, designed specially to be used in endoscopy, was presented before the Southern Medical Association in November 1944. This model is now employed only for foreign bodies lodged in the esophagus and the stomach. For the removal of these it is fixed to a Levin tube, size 12 F. A metallic stylet in the tube increases the control of its motility. A small opening in the tube at the magnet end and a bulb bellows at the other enable one to inflate the stomach in order to allow free play of the foreign body and to improve the maneuverability of the magnet. The intragastric use of such a magnet should all but obviate the necessity of cutting through the abdominal wall into the stomach to remove magnetizable foreign bodies.

A second model of the magnet for intratracheal and intrabronchial use, a cylinder 4 cm. in length and 0.3 cm. in diameter, was announced

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From the Ponce de Leon Ear, Nose and Throat Infirmary.

Presented at the meeting of the American Laryngological, Rhinological and Otological Society, Inc., Chicago, May 27, 1946.

in March 1945. This model is attached to a ureteral catheter for intrabronchial work.

A number of cases in which an open safety pin was removed by means of the Alnico magnet have already been reported. If the pin is in the esophagus with the point up, and it always is when in the esophagus, the magnet, introduced through a laryngoscope, is used to pull the pin down into the stomach. When the foreign body is in the stomach, the stomach is inflated and the Levin tube is manipulated until there is a complete reversal of the position of the pin with respect to the magnet. Once this is effected, the magnet is rapidly withdrawn, bringing the pin with it, point down. The trailing point causes no damage to the lining of the esophagus.

In order to improve the fluoroscopic guidance so necessary in this type of work and to simplify the taking of films, a frame to support the patient like that used in various bronchoscopic clinics for a number of years has been modified by substituting a plastic plate for the top. It has been further modified by tunneling under the plate so as to permit a casset to be inserted from the side. With this arrangement, it is possible to take a film with a radiographic tube 36 inches (91.5 cm.) above the casset.

Formerly, when an open safety pin had lodged in the esophagus, the surgeon had to grasp it with forceps and push it down into the stomach, where there was difficulty in reversing the pin as well as danger of perforation. An alternative was to close the pin in the esophagus; but this could rarely be done, and when accomplished it resulted in considerable trauma of the organ. Even in the most expert hands, some of the children who swallowed open safety pins died. It is in this field, therefore, that the magnet has proved of greatest use; it converts a difficult operation with a fairly high mortality into a simple one almost devoid of danger. Because of the frequency with which babies manage to swallow open safety pins, I should like to record my recent case:

An infant of 9 months swallowed an open safety pin (fig. 1 *A*). The next day, April 5, 1946, he was admitted to the Ponce de Leon Ear, Nose and Throat Infirmary. Anesthesia was induced with ether, a laryngoscope was inserted, the magnet was pushed down the esophagus under fluoroscopic guidance, and contact with the pin was established. The pin was then pushed into the inflated stomach (fig. 1 *B*), flipped on the magnet and withdrawn (fig. 1 *C*). The next day the infant went home well.

Two cases in which the magnet was used for the removal of other foreign bodies may be reported:

An aviator, who had completed without mishap thirty-five missions over Europe during the recent war, was one day picking his teeth with an ordinary straight pin. In some inexplicable way he swallowed the pin. The first roentgeno-

gram showed the pin in the stomach (fig. 2 *A*). While he was being anesthetized, he became panicky, retched and vomited. When he became quiet, to the surprise of those attending him, fluoroscopic examination failed to show the pin in the stomach. Further search finally revealed it in the lower part of the right main

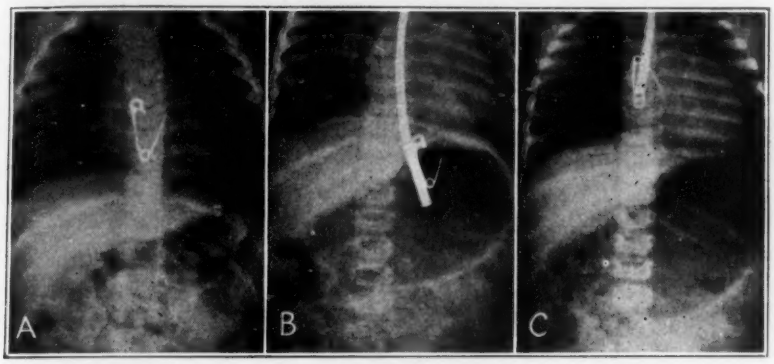

Fig. 1.—*A*, open safety pin lodged in the middle portion of the esophagus. *B*, safety pin being passed downward into the stomach. *C*, reversed pin being drawn upward with point trailing.

stem bronchus, point up (fig. 2 *B*). The bronchoscope was passed down to the right lung with fluoroscopic guidance, and the smaller magnet, instead of the ordinary forceps, was inserted through the bronchoscope and pushed toward the pin. The magnet promptly attracted the pin (fig. 2 *C*), and magnet and pin were then rapidly withdrawn through the scope. In this way there was no trauma of the lung. He was able to leave the hospital two days later, quite recovered.

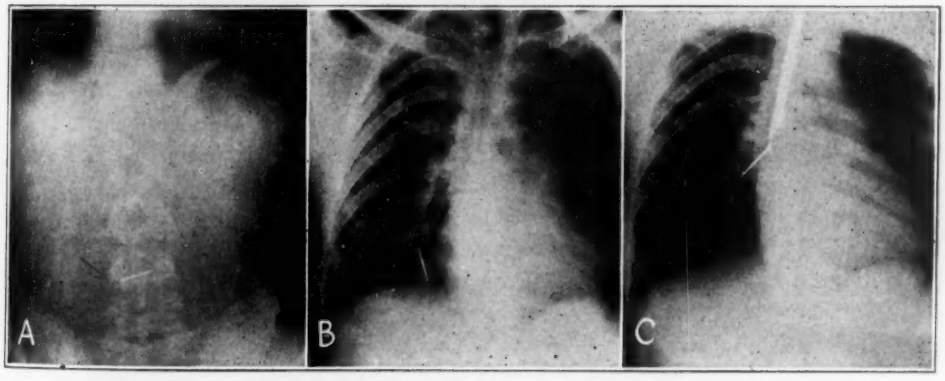

Fig. 2.—*A*, straight pin in stomach (the pin has been retouched for photographic purposes.) *B*, same pin in the right main stem bronchus as a result of regurgitation and aspiration of contents of the stomach. *C*, magnet in contact with the pin, ready to remove it, after having been passed down the trachea and into the main stem bronchus.

A nurse, ill with bronchitis, held several bobby pins in her mouth while rolling up her hair before going to sleep. Following a cough, she swallowed not one but

three of the pins (fig. 3 *A*). By means of the magnet, two pins were withdrawn the first time the magnet was introduced into the stomach (fig. 3 *B*), and the third was located a few seconds later and removed with equal ease.

It is appropriate here to voice a word of caution with regard to foreign bodies lodged in the stomach. It has long been recognized that most things that can enter the stomach through the natural channels without harming them can pass out through distal channels equally harmlessly. Before the days of the magnet the removal of intragastric foreign bodies was exceedingly difficult. Therefore when such an object had entirely entered the stomach, one had the choice of attempting to remove it with Tucker's flexible forceps under fluoroscopic or gastroscopic guidance, performing a laparotomy or waiting watchfully. The use of Tucker's flexible forceps requires a great deal of experience in

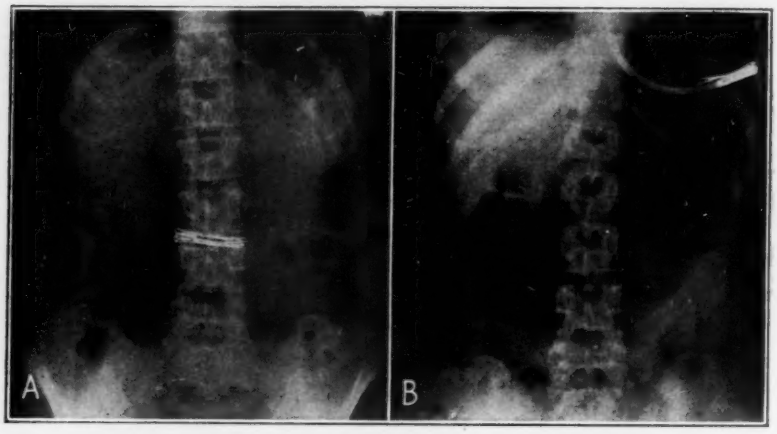

Fig. 3.—*A*, three bobby pins in the stomach. *B*, magnet in contact with the bobby pins just prior to removal.

addition to skill. Laparotomy always carries some risk, and therefore it was usually justifiable to be conservative. The disadvantages of the conservative policy, however, were brought vividly home to me recently: A small child, who had swallowed a bobby pin three days earlier, was admitted to the infirmary. The pin had already entered the duodenum. Two days after admission symptoms of obstruction of the upper part of the intestinal tract developed, and a major operation was necessary to save her life. Remember then, aside from the danger that a sharp or pointed object will perforate the intestinal wall, a long metallic one offers dangers of obstruction until it has passed.

SUMMARY

The Alnico magnet greatly simplifies the problem of removing ferrous foreign bodies, such as pins, safety pins, staples, screws, nails, tacks

and zipper fragments, from food and air passages. The magnet is small enough to be passed easily through a 4 mm. bronchoscope. It requires no wires or batteries. The magnet is of no help when vegetable matter or articles of silver, copper or plastic have to be extracted from the esophagus, the lungs or the stomach.

In removing metallic foreign bodies from the lungs difficulty is often experienced in grasping with forceps an elusive object, such as various types of pins, especially when the point is up. There is always danger of trauma with perforation of the bronchial wall as a result of manipulation.

With the stomach inflated, the removing of a ferrous foreign body from it by means of the magnet is so satisfactory that it should obviate any major surgical operation in almost every case. It is such a simple procedure that one is justified in removing even those objects that would almost certainly pass through the natural channels, if only to spare the patient, or more often the patient's mother, hours of watchful waiting and anxiety. It is in most cases safer, rather than to wait in hope that the foreign body will pass, with the attendant danger of perforation and obstruction, to insert the magnet, withdraw the offending object and send the child home the next day.

144 Ponce de Leon Avenue.

Case Reports

OTOSCLEROSIS AND BLUE SCLERAS

DANIEL W. BRICKLEY JR., M.D.*
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THE STRIKING clinical syndrome of blue scleras, brittle bones and otosclerosis has been accorded little attention by otologists in recent years. More reports have been made concerning the orthopedic and the ophthalmic than the otologic features. This may be explained by the fact that the deafness is a manifestation which usually occurs later, in the second or third decade of life, and therefore is less often included in the statistics. A possible clue to the causation of otosclerosis may be found through studies of this interesting and unique syndrome. Two cases studied at one of the Army hearing centers are reported in this article. The condition has been called osteosclerosis fragilis, fragilitas ossium and osteogenesis imperfecta. The cause is unknown, but there is a marked hereditary tendency toward the development of this condition, which all writers on the subject have emphasized.

Henzschel,[1] in 1831, and Gescheidt, in 1832, first described blue scleras. Spurway, in 1896, Burrows, in 1911, Adair-Dighton,[2] in 1912—all described blue scleras and noted a decided tendency toward multiple fractures. Eddows,[3] in 1900, first reported four generations of a family with blue scleras and fragile bones and showed that the syndrome is inherited and follows the mendelian law as a dominant characteristic. He did not include deafness in his description. In all of the series studied, 50 per cent of the offspring of a heterozygotic person had blue scleras and brittle bones. The unaffected children did not have affected descendants. The line of transmission is not through females to males. A generation is never skipped as occasionally happens in hemophilia (knight's move of Bateson).

Bronson,[4] in 1917, reported a careful study of two families. The first family, of four generations, comprised 55 persons; 21 had blue scleras; 20 of this 21 had suffered multiple fractures, and 7 had deafness. The second family, of three generations, comprised 8 persons; 7 had blue scleras, 4 had suffered fractures and none had deafness. Bronson stated that, in addition to the three prominent features of the condition, there was a tendency to be below average height and that the patients spoke of themselves as being small boned in contrast to their relatives who had white scleras. Bronson noted also that the frontal and occipital bones were prominent but that the head was not

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1. Cited by Key.[5]
2. Adair-Dighton, C. A.: Ophthalmoscope **10**:188, 1912.
3. Eddows, R.: Brit. M. J. **2**:222, 1900.
4. Bronson, E.: Edinburgh M. J. **18**:240-281 (April) 1917.

square, as it is in cases of rickets. Key [5] stated that this is a typical osteogenesis imperfecta type of skull. In Bronson's first family there had been 2 members who had patent fontanels throughout life. Later authors have not confirmed the presence of any typical skull formation.

Riddell,[6] in 1940, traced a large pedigree consisting of 208 persons for this condition; 45 had blue scleras. He emphasized the strong hereditary tendency but made no conclusions. He added color blindness to the triad.

Key,[5] in 1926, reported a case and reviewed the literature comprehensively. He stated that the abnormalities seem to be due to defects of development of some of the tissues which arise from the mesenchyme —so-called hereditary hypoplasia of the mesenchyme. This hypoplasia is an anomaly of development rather than a derangement of metabolism.

Riesenman and Yater,[7] in 1941, reported a study of 255 members of seven family groups, with 91 members presenting this syndrome. They stated that fragilitas ossium is hereditary mesenchymal hypoplasia and is transmitted as a dominant mendelian factor.

Corboy [8] stated that 60 per cent of patients with blue scleras have fragile bones, 60 per cent have otosclerosis and 44 per cent have all three defects. He presented the case of a mother, two sons and a daughter with this syndrome; all 4 had low blood calcium levels (7.53 to 8.01 mg.) and low phosphorus levels (1.96 and 2.19 mg.)

Farber and Margulis [9] reported a family of 52 members and gave a genealogic tree. Twelve members had blue scleras; 7 of these had brittle bones, and 4 of the 7 had otosclerosis. They pointed out that the parathyroid glands apparently are not implicated pathogenetically.

Fox and Sweet [10] stated that the blood calcium and phosphorus levels of patients with this syndrome are usually within normal limits, although isolated ones may show a disturbance of calcium metabolism and hyperparathyroidism.

THE INVOLVEMENT OF THE EYES

The abnormality of the eyes is the most prominent and striking feature of the syndrome. A blue appearance of the scleras is the first and the essential part of the clinical picture, because only those members of the family who have blue scleras are subject to the brittle bones and the deafness. The color has been described as clear china blue, slate blue or gray blue. The color is darker in the anterior portion of the sclera over the ciliary body, which can be distinctly outlined.

5. Key, J. A.: Brittle Bones and Blue Sclera: Hereditary Hypoplasia of the Mesenchyme, Arch. Surg. **13**:523-567 (Oct.) 1926.

6. Riddell, W. J. B.: Ann. Eugenics **10**:1-13 (April) 1940.

7. Riesenman, F. R., and Yater, W. M.: Osteogenesis Imperfecta: Its Incidence and Manifestations in Seven Families, Arch. Int. Med. **67**:950-967 (May) 1941.

8. Corboy, P. M.: Am. J. Ophth. **24**:57-60 (Jan.) 1941.

9. Farber, J. E., and Margulis, A. E.: Blue Scleras, Brittle Bones and Deafness: Report of Afflicted Family, Arch. Int. Med. **71**:658-665 (May) 1943.

10. Fox, M. S., and Sweet, S. J.: Brittle Bones Associated with Deafness and Blue Scleras: Report of Syndrome in Two Persons of One Family, Arch. Otolaryng. **32**:505-511 (Sept.) 1940.

The anatomic cause of the blue color is still in dispute, although all authors agree that it is not due to pigmentation but is a result of an abnormal transparency or translucence of the sclerotic coat which permits the blue uvea to shine through. Conlon,[11] in 1913, considered the unusual transparency as probably due to a change in the quality of the tissue and not to thinning of the sclera.

There is no increase in visual defects or refractive errors, nor are there abnormal changes in intraocular tension in these persons. In most of the cases in which refraction was studied, astigmatism and hypermetropia were shown. In one of my cases mild myopic astigmatism was found and in the other, mild hyperopic astigmatism. Embryotoxon is apparently not of great significance in this syndrome.

DEAFNESS

Deafness was first mentioned by Adair-Dighton,[2] in 1912, when he noted its presence in 1 member of a family of four generations with blue scleras. Van der Hoeve,[1] in 1917, first recognized that 60 per cent of the patients have otosclerosis and connected the triad. This syndrome is now usually identified by his name. Also in 1917 de Kleijn [1] and Bronson [4] independently showed that in persons with this syndrome there tended to develop in the third and fourth decade a conduction type of hereditary deafness. The deafness was of otosclerotic origin in all cases, and when the condition was advanced there was superimposed labyrinthine disease with nerve involvement. It was emphasized by van der Hoeve and de Kleijn that in many of the younger patients the beginning deafness had not been noticed but was brought out by aural examination. Ruttin,[12] in 1919, stated that deafness was omitted from the statistics because of its late appearance.

The cause of the deafness was explained by Bronson on the basis of a deposit of calcium salts in the middle ear. Ruttin showed the presence of otosclerosis by histologic examination of the petrous bone, and this has been confirmed in the few cases in which pathologic studies of the temporal bone have been made. Occasionally an excessive amount of calcification of the petrous portion of the temporal bone, presumably of the region of the vestibule, has been noted in roentgenograms. Stenvers made roentgen studies of the skulls of the families of van der Hoeve and de Kleijn and found that the petrous bone cast a dense shadow and that there was a compact opaque mass covering and obscuring the entire vestibular region.

Shambaugh,[13] in a personal communication, reported 2 patients with blue scleras on whom he did the fenestration operation. One had multiple fractures, and the other has a daughter with multiple fractures. Nothing unusual was observed in the labyrinthine capsule at operation, but the mastoid cell partitions were unusually delicate and brittle. In each patient the operation resulted in an excellent improvement in hearing that was sustained. These are the only cases of this type in which the fenestration operation has been done. Perhaps in the future operation on persons with this syndrome will shed more light on the

11. Conlon, F. A.: Boston M. & S. J. **169**:16-18, 1913.
12. Ruttin: Monatschr. f. Ohrenh. **54**:305, 1919; cited by Key.[5]
13. Shambaugh, G. E., Jr.: Personal communication to the author.

pathology of otosclerosis. The deafness is of the conduction type until the condition is advanced, when nerve involvement can often be demonstrated. It fulfils all the criteria of clinical otosclerosis—a family history of deafness; no prior history of aural disease; onset in the third and fourth decades of life; a pink reflex over the promontory; patency of the eustachian tubes; audiometric loss for both high and low tones; bone conduction usually normal or only slightly impaired; air conduction greatly impaired; Rinne test negative; audiogram showing typically a greater loss in the low frequencies by air conduction and less loss in the frequencies above 1024 double vibrations.

FRACTURES

Multiple fractures of the long bones are present in 60 to 70 per cent of patients with blue scleras. This condition has received much attention in orthopedic literature and only a brief review need be made here. The fractures are not spontaneous, but less violence is necessary than to break a normal bone. There is often less pain with a fracture, possibly because there is less trauma. The healing is not delayed and may be even faster than that of normal bone. There is frequent dislocation of joints, owing to the relaxed condition of the ligaments and surrounding soft tissue. Fractures and dislocations are common in childhood and tend to diminish after puberty. Roentgenograms of the skeleton show a general decrease in size of the bones and concentric atrophy of the long bones. The bones are unusually hard and brittle; the lamellas are irregular, and the amount of compact bone is decreased by the presence of numerous large canals. As stated earlier, Key [5] explained the brittleness of the bones on the basis of hypoplasia of the mesenchyme.

REPORT OF CASES

Case 1.—A 22 year old white man was admitted to the hospital May 6, 1945. He first noted the onset of a hearing loss associated with tinnitus aurium in April 1942. He had undergone considerable acoustic trauma as engineer gunner and as armament line chief in the Army Air Forces where he was exposed to the noise of 50 caliber machine gun fire and the roar of airplane motors. The hearing loss had been progressive since the onset in April 1942. He knew of his China blue scleras, which he had had since birth. From the age of 3 to that of 16 years he had suffered multiple fractures, involving both elbows, the left forearm, the left thumb, the left leg and the right foot, which resulted in a mild deformity of the left thumb and the left elbow and limited extension of the right elbow. The accidents resulting in these multiple fractures were various and included falls from a bicycle, slipping on a banana peel, falling down stairs, and some experienced while he was engaged in sports. The fingers of both hands had been double jointed, limber and hyperextensible since birth. His teeth had been in excellent condition and did not demonstrate any caries until the past year.

As to the family history, in generation 1 the paternal great uncle had brittle bones, blue scleras and deafness. One boy and one girl in this generation did not manifest the disease. In generation 2 the patient's father and a paternal aunt have brittle bones, blue scleras and deafness. Generation 3 includes the patient as one of ten children; one brother, aged 14, has a combination of brittle bones, deafness and blue scleras; one sister, aged 13, and another aged 15 months, have blue scleras and no other stigmas; the other members of the family are apparently free of the condition.

At the time of examination the patient was a fairly well developed white man of average height (66 inches [167.5 cm.]) and weight (142 pounds [64 Kg.]) who showed the skeletal abnormalities mentioned and also mild left scoliosis of the lower dorsal region of the spine. The eyes showed the typical blue scleras; the fundi were normal. Vision was 20/15 bilaterally, and refraction showed mild compound hyperopic astigmatism. The nasal septum was irregular, and the posterior tip of the left inferior turbinate was hypertrophied. The nasopharynx was clean; the tonsils had been removed in 1940. The tympanic membranes were intact and appeared normal. The eustachian tubes were patent.

The blood was normal, and the Kahn test of the blood was negative.

Red Blood Cells	Hemoglobin, Gm. per 100 Cc.	White Blood Cells	Neutrophils, per Cent	Lympho-cytes, per Cent	Blood Calcium, Mg. per 100 Cc.	Blood Phosphorus, Mg. per 100 Cc.
4,600,000	14.8	6,800	72	28	9.7	3.3

The urinalyses gave normal results. Roentgenographically, the long bones were normal, and also the chest and the mastoid processes.

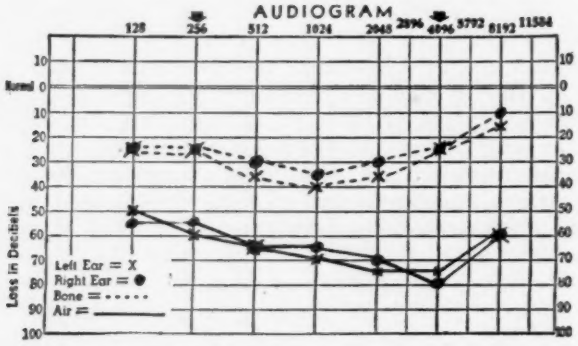


Chart 1 (case 1).—Audiogram of a 22 year old white man who had blue scleras, a history of multiple fractures and progressive loss of hearing.
Speech test: 0/20 right ear; 0/20 left ear.
Whisper test: 0/15 right ear; 0/15 left ear.
Average loss in decibels—
Air conduction: right 67; left 70.
Bone conduction: right 32; left 37.
Percentage loss: right 54; left 56.

Hearing Program.—With the 256 double vibration fork, the Rinne test was negative bilaterally, and the Schwabach test showed hearing decreased bilaterally. Hearing for low conversational speech and for whispering was 0/20 and 0/15 bilaterally, respectively. The audiogram showed a curve typical of advanced otosclerosis with some involvement of the cochleas. The audiometric loss in the speech frequency range was: right ear 67 decibels for air and 32 decibels for bone conduction; left ear 70 decibels for air and 37 decibles for bone conduction. This nerve involvement was interpreted as being the result of an otosclerotic process progressing normally into the cochlea and not that of nerve damage due to acoustic trauma. It is felt that otosclerotic ankylosis of the stapes is probably the best insulation against nerve damage due to acoustic trauma. He received four weeks of auditory training, eight weeks of lip reading and four weeks of speech insurance; he was fitted with an air conduction hearing aid for the right ear, with which he obtained good hearing to 20 feet (6 meters).

CASE 2.—A 31 year old white man was admitted to the hospital March 26, 1946. He first noted hearing loss bilaterally in May 1944 at the age of 29 years, and it had been slowly progressing since that time. He had had blue scleras since birth and had had multiple fractures since the age of 5 years, when he broke his right leg falling down stairs. He broke his left leg in 1920, at the age of 6, when he fell out of a hammock. The right wrist was broken in 1924, the right elbow in 1928. He sprained his back in 1929 in a fall and could not walk for three months. He fractured the left clavicle in 1932. The left shoulder was broken in October 1932, when he was struck by an automobile. It is interesting to note that in the latter accident he suffered a penetrating wound of the left eye which required enucleation of the eye. Inquiry at the hospital where the enucleation was done showed that unfortunately the eye was not examined histologically. He stated that he fractured some ribs in 1939, at the age of 25, while wrestling, and that he fractured the fifth finger of the left hand in March 1945.

The family history indicates that the defect is carried on the maternal side of the patient's family. In generation 1 the maternal grandmother had blue scleras, but it is not known whether she had any loss of hearing. In generation 2 the mother has blue scleras, has been hard of hearing since she was a young girl and has an indefinite history of fracture of bones occurring in adolescence. Four maternal aunts and one maternal uncle have blue scleras; nothing is known of fractures and deafness among these relatives. The patient stated that some of his maternal cousins also had blue scleras and that some did not. Generation 3 includes the patient as one of five boys; four have blue scleras and a history of multiple fractures, and one has had no broken bones and his scleras are white. The patient stated that he is the only one who has a hearing loss in this generation. He has one nephew (generation 4) who is 16 years of age at the present time, has blue scleras and has very small bones, is of small stature and for this reason would like to follow the profession of a jockey; however, he has suffered several fractures of the long bones and has had to give up this ambition.

At the time of examination the patient was a well developed and well nourished white man, 67 inches (170 cm.) in height and weighing 145 pounds (65.5 Kg.). The extremities tended to be slender, and there were residuals of fracture of the right elbow and the fifth finger of the left hand. There were mild lordosis and scoliosis of the spine. There was no evidence of dental caries. The right eye revealed a deep blue sclera, and the left eye was artificial. Vision of the right eye was 20/30, refraction showed compound myopic astigmatism, and vision was corrected to 20/20 with: -1.25 D. sph. $\bigcirc +$ 0.75 D. cyl., ax. 145 The fundus was normal. The nasal septum was irregular and the airway adequate. The nasopharynx was clean, and the tonsils were not remarkable. The right tympanic membrane was intact, dull, retracted, and there was a healed perforation anterior to the umbo. The left tympanic membrane was intact, dull, retracted, and there were two healed perforations covered with a thin, hypermobile membrane, although the patient said there had been no aural suppuration. The eustachian tubes were patent.

Routine studies of the blood, including determinations of calcium and phosphorus and the Kahn test, gave normal results. The urinalysis gave normal results.

Red Blood Cells	Hemoglobin, Gm. per 100 Cc.	White Blood Cells	Neutrophils, per Cent	Lymphocytes, per Cent	Monocytes, per Cent	Eosinophils, per Cent	Blood Calcium, Mg. per 100 Cc.	Blood Phosphorus, Mg. per 100 Cc.
4,340,000	15	8,200	69	26	3	2	9.5	3.3

The radiologist reported that roentgenograms of the long bones did not have the typical appearance that one would expect in a case of blue scleras. Roentgenograms of the lungs and the mastoid processes were normal.

In 1939 the patient had gonorrheal urethritis, and the Wassermann test of the blood was positive at that time. He received arsenical and bismuth therapy for eighteen months, and on induction in October 1943 an examination of the spinal fluid was negative for syphilis.

Hearing Program.—With the 256 double vibration fork the Rinne test gave a negative and the Schwabach test a normal result bilaterally. Hearing for low conversational speech was 0/20 bilaterally and that for whispered voice 0/15 bilaterally. The audiogram showed a typical conduction type of deafness: right ear, a 50 decibel loss for air conduction and a 7 decibel loss for bone conduction in the speech frequency range; left ear, a 55 decibel loss for air conduction and a 7 decibel loss for bone conduction in the same range. The patient received four weeks of auditory training and six weeks of lip reading. He was fitted with an air conduction hearing aid for the left ear, with which he had good hearing to 20 feet.

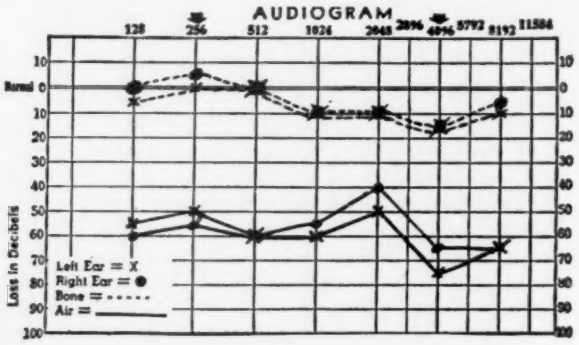


Chart 2 (case 2).—Audiogram of a 31 year old man with blue scleras, a history of multiple fractures and progressive loss of hearing.
Speech test: 0/20 right ear; 0/20 left ear.
Whisper test: 0/15 right ear; 0/15 left ear.
Average loss in decibels—
Air conduction: right 50; left 55.
Bone conduction: right 7; left 7.
Percentage loss: right 40; left 44.

SUMMARY

The clinical syndrome of blue scleras, fragile bones and otosclerosis is described briefly. Two cases are presented, illustrating the factor of heredity; in one the transmission was through the male side and in the other through the female side. In the first case the deafness was initially manifested at the age of 19 years and showed rather rapid progression, with the cochleas becoming involved in the relatively short time of three years. In the second case the deafness was noted at the age of 29 years and there was no nerve involvement. In each case an air conduction hearing aid produced good hearing to 20 feet (6 meters).

115 North Prospect Street.

TRAUMATIC DISSECTION OF THE ESOPHAGUS; MEDIASTINITIS WITH RECOVERY

JAMES E. DAVIS, M.D., HARTFORD, CONN.

THE TREATMENT of injuries and infections of the mediastinum always presents a difficult problem. Before the advent of sulfonamide compounds and antibiotic drugs the mortality rate was high, but since the use of these therapeutic aids has become standard treatment in infections the rate has decreased remarkably. Even with these helps and with the advances that have been made in thoracic surgery such injuries and infections still at times present serious problems, and prognosis is always guarded. A case of an unusual type of mediastinal injury with subsequent infection is reported here, which demonstrates the value of sulfonamide compounds and penicillin in treatment.

REPORT OF A CASE

H. G., a 9 year old girl, was admitted to the Accident Room of the Hartford Hospital on the evening of Aug. 7, 1945 with a history of having been knocked from her bicycle by an automobile. At the time of admission she was in severe shock. She was unconscious, the skin was moist and cold, the pulse rapid, the respirations labored. The blood pressure was 85 systolic and 40 diastolic. Her temperature was 104 F. There were numerous abrasions and contusions over her body without evidence of fracture of any bones. On the left side of her neck, just anterior to the sternocleidomastoid muscle, about midway between the angle of the mandible and the clavicle there were two ragged wounds from which blood, saliva and air escaped with each respiration. Examination of the chest did not reveal any evidence of pneumothorax or of fluid in the pleural cavities. The lungs were filled with moist rales. Rapid examination of the larynx with a direct laryngoscope revealed that the wounds noted on the external surface had penetrated into the hypopharynx just about the level of the larynx. The larynx and the vocal cords showed no injury. Her condition was so critical that further examination of the tracheobronchial tree and the esophagus was not made, and no attempt was made to clean or close the external wounds. Shock treatment was begun, oxygen was given intranasally, and the pharynx was kept as free as possible of fluid by maintaining suction with an intranasal catheter. Intramuscular injection of penicillin was immediately started, 25,000 units being given every three hours, and a prophylactic injection of tetanus antitoxin was given.

The following day the patient showed periods of mental clearing of short duration but did not respond to questioning. Her temperature remained elevated, and the skin was now dry and hot and the face pale and anxious. A white blood cell count showed 21,500 leukocytes per cubic millimeter, 96 per cent of which were polymorphonuclear cells. A roentgenogram of her chest revealed extensive subcutaneous emphysema of the neck and thorax, with air extending between the muscle sheaths and into the mediastinum. The mediastinal space appeared

widened, and air was noted along the anterior aspect of the chest, apparently extrapleural, and chiefly below the sternum, extending downward into the epigastric region. In both pulmonary fields, particularly in that of the upper lobe of the right lung and the left central region, were areas of increased density due either to small sites of lobular atelectasis or to intrapulmonary hemorrhage. During the afternoon the patient began to show evidence of respiratory distress with marked epigastric and intercostal retraction. She was given 750 cc. of isotonic solution of sodium chloride intravenously and taken to the operating room, where tracheotomy was done, with immediate relief. A further examination of the hypopharynx was made at this time and showed considerable edema of the arytenoids and the false cords, enough to interfere with respiration, but no definite injury of the laryngeal tissues was noted. There were seen, however, a wound which extended down along the left side of the esophagus almost to the diaphragm and a similar wound along the right side of the esophagus. The proximal part of the esophagus was attached anteriorly and posteriorly, but the esophageal mouth was wide open, owing to the absence of the cricopharyngeal muscle, which had been lacerated by the injury. I inserted a small esophagoscope and could find no injury of the esophageal wall; there appeared to be attachment anteriorly and posteriorly along the whole tube. It was apparent from this examination that she would be unable to take any nourishment by mouth for an indefinite period. The routine intravenous injection of dextrose-saline solution was started, 500 cc. being given every twelve hours, plus 250 cc. of citrated blood every twenty-four hours, with 1.5 Gm. of sulfadiazine in each intravenous feeding.

The day following tracheotomy she had regained consciousness sufficiently to ask for water and was no longer incontinent. Her mental condition gradually improved, and at the end of a week her mind was perfectly clear. On August 13, six days after her admission, pharynx, larynx and esophagus were again examined. The lacerations along each side of the esophagus were still open and showed a dirty gray color with a thin serous discharge. The pharyngeal wounds were healing, and air was no longer escaping through the neck. The mouth of the esophagus was still wide open. I again carefully examined the lumen of the esophagus and, finding no evidence of injury, passed a Levine tube through the nose and into the esophagus under direct vision. Feedings were started by this route, and as soon as it was determined that she could take sufficient nourishment through the tube, the intravenous feedings and transfusions were omitted. She was given large amounts of fluids, fruit juices and gruel, as well as adequate vitamins plus 1 Gm. of sulfadiazine every four hours. Up until this time she had continued to have a fever, with temperature ranging from 102 to 104 F. Within forty-eight hours after the feeding tube had been inserted and adequate doses of sulfadiazine given the temperature dropped, and during the remainder of her hospitalization it did not rise above 100 rectally. Her blood, which had been checked every second day since her admission, had shown a gradual drop in white blood cells to approximately normal, and the number remained normal until her discharge. Daily urinalyses had given entirely normal results. On August 14 roentgen examination of the chest showed that the subcutaneous emphysema had almost completely disappeared, but there was still some widening of the superior mediastinum.

On August 21 roentgen examination of the chest was repeated and showed complete disappearance of the subcutaneous emphysema and normal width of the superior mediastinum. At this time she was allowed to be out of bed and was able to walk to the bathroom. She was coughing little, and there was little discharge coming through the tracheotomy tube. This tube was removed on the

sixteenth day after her injury, and healing of the tracheotomy wound proceeded without any complications. On August 25 reexamination of the larynx, the hypopharynx and the esophagus revealed that the edema around the arytenoids had entirely disappeared, and the wounds in the hypopharynx and on each side of the esophagus appeared to be completely closed. The feeding tube was removed. The mouth of the esophagus was still wide open, and there was no closing of the esophagus on swallowing. The external wounds in the neck were healing satisfactorily. The administration of penicillin and sulfadiazine was discontinued, the total doses having been 2,850,000 units of penicillin and 70 Gm. of sulfadiazine. After the feeding tube was removed, as soon as it was determined that she could swallow without discomfort or difficulty she was given increasing amounts of soft foods and within a short time was able to take a regular diet. During the next ten days her progress was entirely satisfactory, but at that time she contracted a mild infection of the upper respiratory tract, which persisted for a week. On September 17 examination of the pharynx and the esophagus was repeated. There was still no sphincteric action at the mouth of the esophagus, but there was no evidence of esophageal stricture. The tracheotomy wound and the wounds of the left side of the neck were completely healed. She was discharged from the hospital on her forty-second hospital day with all wounds completely healed. Her general physical condition was excellent. She had regained her normal weight, and her blood count was essentially normal except for very mild secondary anemia.

She returned to the hospital in the latter part of November 1945 for reexamination, which showed no acute inflammation of either the pharynx or the larynx. The esophageal mouth was still wide open, and no action of the constrictor muscles were apparent. The lumen of the esophagus was not narrowed, and the mucous membranes were normal. She was able to swallow without difficulty and was in excellent condition. She returned to school in January 1946 and was not seen again until August 1946, at which time she appeared to be in perfect health. Examination gave entirely negative results except for the fact that the sphincteric action at the mouth of the esophagus was absent. This loss of function apparently interferes in no way with her ability to take food, and it seems reasonable after a year to assume that no difficulty will arise.

179 Allyn Street.

SCHWANNOMA OF THE FACE

MARK THUMIM, M.D., MIDDLETOWN, CONN.

THIS REPORT is being made because of the rarity of schwannoma (tumor of the sheath of a peripheral nerve) in the region of the nasolabial fold. Stout [1] stated that the distribution of schwannoma is not even. In some sites it is more commonly seen, the stomach, the neck and the extremities being among these, and in some sites it is seen rarely. Among the 246 cases (50 of his own) which he found recorded in the literature, there were few in which the tumor occurred in the region of the nasolabial fold—in 1, on the upper lip; in 1, in the ala nasi; in 1, lateral to the angle of the mouth. Ehrlich and Martin [2] reviewed the cases observed at Memorial Hospital for the Treatment of Cancer and Allied Diseases, New York, from 1936 through 1942, when new cases of tumors of the head and neck averaged 1,600 yearly. They reported a case of schwannoma of this area as "swelling over the nose." These authors included a complete review of the disease, and there is no need for repetition. No other reports were found.

REPORT OF A CASE

A 21 year old white man noticed a small swelling just to the right of his nose about four months before examination. The mass had been getting larger. There was no history of trauma, nor had he experienced any pain. He had had some difficulty in breathing through the right side of the nose.

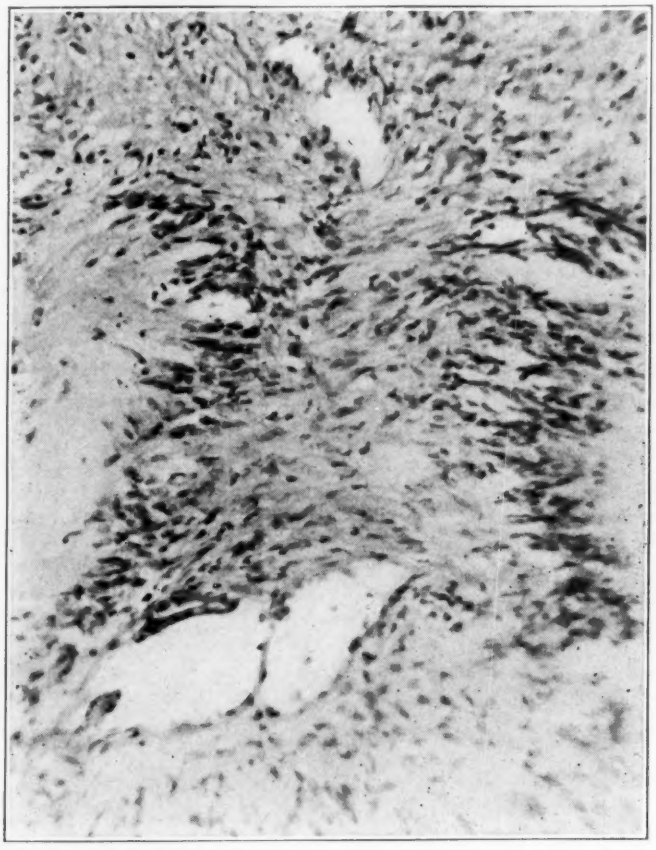
On examination there was a soft mass in the tissue of the face. It made a swelling, the most prominent part of which lay just above and parallel to the right nasolabial fold. The right ala nasi was thick and firm. No other masses were seen on the body. Roentgen examination showed no bone involvement and normal sinuses. At operation the mass was encapsulated, soft, friable, lobulated, gray.

Laboratory Report (Dr. Morris Goldenberg).—"The specimen consists of two irregular pieces of yellow-tan tissue. The larger measures 3.0 by 1.0 by 0.8 cm. and has the appearance of irregular strands of adipose tissue connected together. The consistency, however, is more firm, and the surface is somewhat granular and papilliferous. The cut surface is yellow-gray with reddish brown streaks. The smaller piece measures roughly 1.5 by 1.0 by 0.7 cm. The general appearance and the consistency are the same. Microscopically (figure), multiple sections from two blocks show a similar picture of a cellular tumor composed of thin spindle-shaped and oval nuclei with cell outlines which cannot be made out

1. Stout, A. P.: The Peripheral Manifestations of the Specific Nerve Sheath Tumor, Am. J. Cancer **24**:751, 1935.

2. Ehrlich, H. E., and Martin, H.: Schwannomas (Neurilemomas) in the Head and Neck, Surg., Gynec. & Obst. **76**:577, 1943.

and with cytoplasmic fibrils which fuse and anastomose. The characteristic features are the large whorl arrangement and the palisading of the nuclei in pronounced parallel pattern. Some areas are notably cellular, and here the nuclei are larger, plump and oval and more chromatic, but no hyperchromatism is noted, and only one mitotic figure has been found after prolonged search. Small acellular areas consist of a uniform, almost hyalin-like tissue, which in places is undergoing myxomatous degeneration which stains poorly and loses the fine



Photomicrograph of a section of schwannoma of the nasolabial fold; × 250.

fibrillar structure seen elsewhere. There are small and moderate-sized hemorrhages and foci of a golden brown granular pigment, hemosiderin. Several small cysts are seen. Diagnosis: neurilemoma."

COMMENT

This case is reported because of the rarity of this tumor in this location.

57 South Main Street.

Clinical Notes; New Instruments and Technics

A PRESSURE SPLINT WHICH AIDS IN CLOSING AN ORAL ANTRAL-ALVEOLAR FISTULA

J. MATHEWS ROBISON, M.D.
Houston, Texas

THIS APPLIANCE was devised by one of my patients, Mr. George Weaver. He had had three unsuccessful operations for closure of an oral antral-alveolar fistula before consulting me. I operated on him by joining palatal and buccal flaps, but the fistula failed to remain closed. Following a second similar operation, a pressure bandage was applied to his cheek in the region of the fistula. The bandage was cumbersome, difficult to hold in place and interfered with his work. He devised the appliance illustrated here and wore it constantly for three weeks; I am sure it was instrumental in aiding the closure of the fistula. Three months later he had three upper molar teeth extracted on the opposite side. A large fistula resulted, which closed after one operation and the use of this appliance. Apparently this device immobilizes the buccal area, aids in approximation of the flap used in closure and reduces the tension on the sutures.

The wire used was obtained from a wire coat hanger, and the rubber bands were cut from an automobile inner tube.

1304 Walker Avenue.

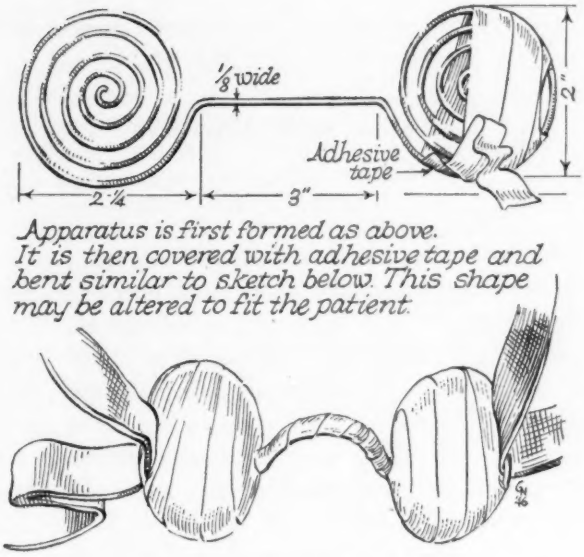

1/8 wide
2"
Adhesive tape
2 1/4
3"
Apparatus is first formed as above.
It is then covered with adhesive tape and
bent similar to sketch below. This shape
may be altered to fit the patient.

Fig. 1.—Diagram of the splint.

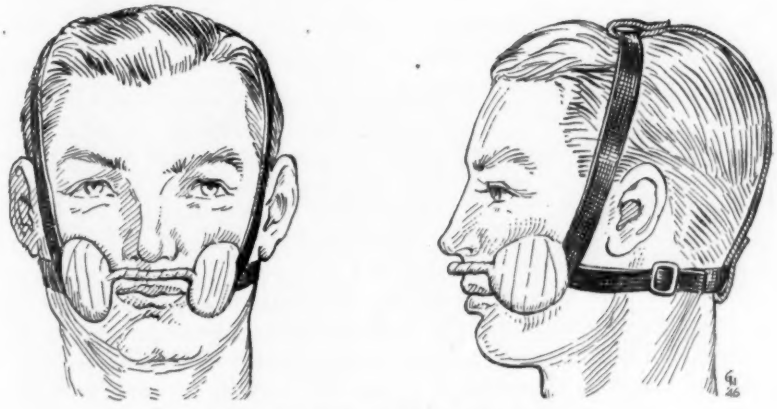


Fig. 2.—Application of the splint.

Progress in Otolaryngology

Summaries of the American and British Bibliographic Material Available in the Field of Otolaryngology

TONSILS AND ADENOIDS

J. DUDLEY SINGLETON, M.D.
DALLAS, TEXAS

A REVIEW of the American and British literature of 1945 reveals several worth while articles. A large number of the contributions are from men who were serving in the armed forces. Chemotherapy received a great deal of attention. The beneficial effects of penicillin as used in the treatment of acute tonsillitis and pharyngitis was the most popular subject. There were a few reports on the use of the sulfonamide drugs. Reports of the efficacy of bismuth as used in the treatment of acute tonsillar and pharyngeal infections also appeared. A few articles dealt with postoperative bleeding at the sites of tonsillectomy and adenoidectomy. Methods of inducing anesthesia for tonsillectomy received some attention from various authors.

TONSILS—GENERAL CONSIDERATIONS

The Commission on Acute Respiratory Diseases[1] reports on an exhaustive study of a food-borne epidemic of tonsillitis and pharyngitis due to beta hemolytic streptococci, type 5. The article is well summarized by the authors as follows:

A food-borne epidemic of tonsillitis and pharyngitis caused by β-hemolytic streptococci of Group A, type 5, was studied. Epidemiological evidence incriminated creamed eggs served at breakfast as the probable vehicle of infection. The outbreak was explosive in onset, with a median incubation period of 38 hours and a primary attack rate of 42 per cent. Almost all subjects originally infected became critically ill. The secondary attack rate was 30 per cent, of which half were cases and half carriers.

The illnesses produced by epidemic type 5 streptococcus infection were mild to moderately severe and did not differ appreciably from sporadic contact, or airborne, cases of tonsillitis and pharyngitis caused by other types of streptococci at this Post. As in previous studies of sporadic streptococcal infection, the history and appearance of the throat were typical in the majority of cases but in some the diagnosis could not have been made by clinical criteria alone. Pharyngeal or

1. Commission on Acute Respiratory Disease: A Study of a Food-Borne Epidemic of Tonsillitis and Pharyngtis Due to B-Hemolytic Streptococcus Type 5, Bull. Johns Hopkins Hosp. **77**:143-210 (Sept.) 1945.

tonsillar exudate was present in 90 per cent of the cases but in only a quarter was it confluent and in another quarter it was no more than pin-head in amount. Although symptoms, physical signs and fever were rapid in onset, maximal in intensity within 48 hours, and declined rapidly, complete return to normal was frequently protracted. Untreated patients who had had tonsillectomy exhibited somewhat more fever than those with tonsils but other significant or consistent differences in clinical severity between the 2 groups were not found.

Alternate patients were treated with sulfadiazine. By statistical analysis slight reduction was demonstrated in the daily frequency of certain symptoms, particularly sore throat, but no objective evidence of benefit was found with regard to the local inflammatory reaction. Only a slight decrease in the height and duration of fever resulted from sulfadiazine treatment. While chemotherapy effected striking reductions in total and differential leukocyte counts and in the daily prevalence of B-hemolytic streptococci in the throat, the changes were transitory and persisted only during the period of treatment. From the standpoint of practical therapeutics no worthwhile benefit was derived from chemotherapy in this epidemic.

Suppurative complications were few but a considerable number of nonsuppurative complications were detected. These included 3 cases of acute rheumatic fever and 2 others in which that diagnosis was suggested but not established. Four other patients had unexplained febrile relapses of 1 to 4 weeks' duration. In addition 4 other subjects who were symptomatically well were found to have transient electrocardiographic changes of uncertain meaning. In some of the individuals these changes were probably not of pathological significance, but in others the changes may have been indicative of rheumatic myocarditis.

One probable exacerbation of chronic glomerulonephritis was encountered but no examples of acute glomerulonephritis. Transient microscopic hematuria and other urinary abnormalities were detected frequently in the early course of the illnesses. They may have been instances of "focal" nephritis.

A significant rise in titer of antistreptolysin or antifibrinolysin was found during convalescence in 85 per cent of hospitalized cases. Another 12 per cent of cases had a rise in titer of antistreptolysin of less than two tubes. It is probable in many instances that such changes in titer were not due to technical variation; hence an antibody response probably occurred in more than 90 per cent of all cases of type 5 streptococcus infection. Similar antibody responses appeared in nearly half of healthy carriers indicating that a proportion of "healthy" carriers were subclinical cases. Transient carriers did not exhibit an antibody response while those who carried the organisms for more than 10 days did.

The important relationship between the presence or absence of tonsils and streptococcal parasitism was confirmed. Subjects with tonsils frequently had confluent exudate, harbored the organisms in large numbers in early cultures and tended to remain chronic carriers. Subjects whose tonsils had been removed had less exudate, fewer streptococci, and rid themselves of the organisms sooner. Tonsillectomized patients tended to have lower initial levels of antistreptolysin and higher convalescent titers than patients with tonsils.

In those instances in which sulfadiazine suppressed the growth of streptococci to a considerable degree during the first 10 days of illness, an inhibition of antistreptolysin response was demonstrated.

No correlation was found between the severity of illness and either the initial titer of antistreptolysin or the maximum titer during convalescence. However, the termination or persistence of the convalescent carrier state did appear to be related to the height of the antistreptolysin response.

Leathart [2] reviews and briefly discusses the physiology of the tonsil and the adenoid and poses the question: "Is it possible, by correlating what is known of the physiology of the tonsil with physical signs and symptoms usually considered as justifying its removal, to avoid unnecessary operations and select for this treatment only those cases in which permanent improvement is predictable?"

The author states that the function of the nasopharyngeal lymphoid tissue is to pick up organisms from food or air and to grow those to which immunity is needed; this produces toxin, which is absorbed as necessity demands; tonsils and adenoids have important work to do, especially in young children, for in them autoimmunization has only begun, and many future infections are necessary before it is completed; therefore permanent improvement cannot be expected if tonsils and adenoids are removed while immunization is in progress unless they are infected with an organism to which further immunization is impossible.

The usual grounds for the removal of tonsils—sore throats, sepsis and enlargement—are not considered indications for their removal in most cases. Likewise the three main conditions which are thought to justify the removal of adenoids—mouth breathing, recurrent colds and discharging ears—are not considered sufficient evidence of local disease to indicate this operation.

Tuberculous adenitis, rheumatic fever in certain cases, nephritis in certain cases and exophthalmic goiter are listed as indications for operation.

Moore,[3] in discussing the removal of the tonsils of allergic children, states that hypertrophy of the lymphoid tissue of the throat, including tonsils and adenoids, is one of the signs of allergy. He advises against removal of the tonsils of allergic patients unless the tissues are definitely diseased and producing such complications as rheumatism or cardiac or renal disease. The adenoids should be removed if they are sufficiently large to obstruct the orifices of the eustachian tubes. Except in rare cases the author advises against tonsillectomy and adenoidectomy for allergic children under 6 years of age.

Keleman,[4] in a rather lengthy discussion of the palatine tonsil as observed in the aged, states that it is the purpose of the study to approach this question clinically in continuation of two previous reports on the same subject. His summary of the article follows:

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2. Leathart, P. W.: The Role of the Tonsil and Adenoid Mass in the Production of Immunity in Childhood, Practitioner **154**:312-316 (May) 1945.

3. Moore, G. C.: Tonsillectomy in Young Children with Allergy, J. Oklahoma M. A. **38**:238-239 (June) 1945.

4. Keleman, G.: Clinical Observations on the Palatine Tonsil in the Aged, Ann. Otol., Rhin. & Laryng. **54**:421-435 (June) 1945.

Clinical examination of tonsils was performed in 309 patients, 50 men and 50 women in each of the seventh, eighth, and ninth decades of life, and an additional 9 patients whose ages ranged from 90 to 103.

A steady decline in the size of the tonsils in these years was noted, but complete disappearance is rare; in fact, it was less frequent than the presence of large, fully developed tonsils. Characteristic involutional forms are the vertical narrow ridge, the large drop in the lower pole of the fossa where the greatest part of the remnants are found, and the small crater surrounding the last crypt.

The tonsillar fossa is not included in the involutionary process. It remains a more or less spacious niche with its arches even more conspicuous when empty.

Local inflammations, tonsillar and peritonsillar, are infrequent and take the same course as in any other age group. The attack may be the first one and may involve small remnants or regenerates after tonsillectomy. Fifteen patients with previous removal of the tonsils gave uncomplicated operative histories; the local results were excellent although the improvement in their general condition was dubious. No greater operative risk was present in the aged than in the young.

Around the sixth decade there is a period of high tonsillar activity; regression seems to cease with even a slight possible temporary enlargement. This means that there is a characteristic period of increased tonsillar activity at the onset of senescence corresponding to the similar period before puberty.

Many difficulties in advanced age may be ascribed to the tonsils. With the recognition of their presence even in the highest age groups, preventive geriatrics should observe these organs carefully especially at the threshold of the regressive years.

The article contains twenty references.

Gover and Yaukey [5] report a series of studies of the tonsils based on physical findings in members of low income farm families in the United States. The studies were made during the period extending from November 1939 through November 1940. The tonsils were recorded as normal, slightly diseased, markedly diseased or absent. There is a marked variation in the recorded results of different examiners with respect to the prevalence of diseased tonsils and the percentages undergoing tonsillectomies. The prevalence in persons less than 15 years of age in separate localities varies from 5 to 24 per cent for markedly diseased tonsils and from 11 to 75 per cent for slightly diseased tonsils. The percentages of members of these low income farm families who had undergone tonsillectomies were low; only 5 per cent of white persons less than 15 years of age and 12 per cent of persons 15 to 44 years of age had had their tonsils removed. Males and females showed similar prevalence rates for diseased tonsils. White persons and Negroes showed little variation of these rates.

Bates [6] quotes Ewing's definition of a mixed tumor—"a complex embryonal tumor of local origin which reproduces the normal develop-

5. Gover, M., and Yaukey, J. B.: Physical Impairments of Members of Low Income Farm Families: Defective Tonsils and Adenoids, Pub. Health Rep. **60**: 693-713 (June 22) 1945.

6. Bates, C. E. H.: Mixed Salivary Tumor in the Right Tonsil Fossa with Narcolepsy and Cataplexy, Ann. Otol., Rhin. & Laryng. **54**:812-817 (Dec.) 1945.

ment of the tissues and organs of the affected part"—and reports a case of mixed tumor of the tonsillar fossa associated with narcolepsy and cataplexy. The tumor was removed surgically, and the pathologic diagnosis was: "mixed tumor of salivary or mucous gland." Removal of the tumor resulted in disappearance of the narcolepsy and cataplexy. The author has no explanation for the association of the tumor with these neurologic symptoms.

Killinger [7] describes and illustrates a modification of the Tyding tonsil-seizing forceps and the Tivnen forceps. The modification consists in the attachment of the blade of a tongue depressor to the under side of the lower shank of the grasping instrument, thus enabling the operator to depress the tongue and secure a better view while grasping the tonsil.

Fabricant [8] reviews and summarizes the recent literature concerning tonsils and adenoids under three headings as follows: "Medico-Surgical Aspects," "Relation of Tonsillectomy to Poliomyelitis" and "Radiation in Impaired Hearing."

The article is well written and contains twenty references.

Poliomyelitis.—Anderson [9] reports the results of a survey of the relationship between recent tonsillectomy and bulbar or bulbospinal poliomyelitis made during the epidemic of poliomyelitis that occurred in Utah in the summer of 1943. He found that 43 per cent of the cases of bulbar and bulbospinal poliomyelitis that developed during the worst part of the epidemic, which included the months of July, August and September, occurred in children who had undergone tonsillectomy within thirty days of the onset. He also found that the incidence of poliomyelitis was 2.6 times greater in recently tonsillectomized children than in the general child population and that the incidence of the bulbar and bulbospinal type of the disease was 16 times greater in recently tonsillectomized children than in the general child population.

Kessel and Moore [10] call attention to the fact that it has become common-place to recover the virus of poliomyelitis from the stools of patients with active infection and those of asymptomatic carriers during epidemics and that the virus has also been recovered from "tonsil-adenoid tissue" in 3 of 6 attempts during epidemics.

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7. Killinger, R. R.: A Modification of the Tyding and the Tivnen Tonsil-Seizing Forceps, J. Florida M. A. **32**:137 (Sept.) 1945.

8. Fabricant, N. D.: The Tonsil Adenoid Problem, Am. J. M. Sc. **209**: 542-548 (April) 1945.

9. Anderson, J. A.: Poliomyelitis and Recent Tonsillectomy, J. Pediat. **27**: 68-70 (July) 1945.

10. Kessel, J. F., and Moore, F. J.: The Occurrence of Poliomyelitis Virus in Tonsils and Stools of Noncontacts During an Interepidemic Period, Am. J. Hyg. **41**:25-29 (Jan.) 1945.

In an effort to show during an interepidemic period the incidence of the virus in persons who so far as known had not been in contact with patients having poliomyelitis, the authors began a study in August 1941 which was interrupted in February 1942, resumed in May 1942 and finished in September 1942. In this work monkeys (Macaca mulatta) were used because they can be infected by intracerebral or intranasal inoculation of the virus. The authors summarize and conclude their article as follows:

1. Poliomyelitis virus, of unknown titer, one portion of which was treated with ether and one portion untreated, indicated that:

a. Ether treatment of saline-virus mixtures or of virus-stool emulsions reduced their infectivity.

b. A single intracerebral inoculation of saline-virus mixture of ether-treated virus-stool emulsion is more effective than a single intranasal inoculation of the same material.

c. A single intracerebral inoculation of ether-treated virus was more effective than a single intranasal instillation of the same untreated virus.

2. In a survey involving 136 individuals who had no known contact with clinical poliomyelitis, and from whom both tonsils and stools, pooled in groups of 3 each, were tested for poliomyelitis virus, virus was recovered from 3 tonsil pools and 3 stool pools. One of these tonsil pools and one stool pool were from the same group of 3 individuals. One may therefore conclude that at least 5 of 136 individuals harbored virus either in their tonsils or stools.

3. The survey was performed during an interepidemic period and all but one of the positive findings were obtained during the last 3 months of this period, this one having been encountered during the first month of a major outbreak.

CHEMOTHERAPY

Penicillin.—Plummer and others [11] in a careful, well supervised and worth while study of a small group of 54 patients at the Fort Jay Regional Hospital noted the effects of penicillin as used in the treatment of acute pharyngitis and tonsillitis due to hemolytic streptococci. Only the patients with the more severe forms of these diseases were included; a temperature of 102 F. or above, a characteristic local lesion of redness, follicular swelling, edema and patches of exudate accompanied by severe angina and moderate to severe systemic reactions were present in all. Careful laboratory studies were made before and during the course of study. Cultures of nasopharyngeal material indicated that 45 of the 54 patients were infected with hemolytic streptococci.

The article is well summarized by the authors as follows:

1. Fifty-four patients with severe acute pharyngitis-tonsillitis were studied clinically and bacteriologically. Forty-five of these had cultures positive for group A (Lancefield) hemolytic streptococci and were suitable for making a comparison of different therapeutic measures.

11. Plummer, N., and others: Penicillin Therapy in Hemolytic Streptococcic Pharyngitis and Tonsillitis, J. A. M. A. **127**:369-374 (Feb. 17) 1945.

2. In 28 of the 45 positive cases, penicillin was given intramuscularly in a dosage of 15,000 units every four hours over periods varying from twenty-four hours to six days; sulfadiazine was given in 11 cases and no specific therapy in 6.

3. Penicillin was found to exert a pronounced effect on the number of hemolytic streptococci in the nasopharynx. The culture made twenty-four hours after the start of penicillin was almost always negative, and the serial cultures during the course of therapy remained negative. When penicillin was discontinued, hemolytic streptococci tended to return gradually to the nasopharynx. The frequency of their reappearance in our cases was inversely proportional to the length of time the drug was administered.

4. In 19 patients treated with penicillin for sixty-four hours or more symptomatic relief commenced in eight to twelve hours after the beginning of therapy and was complete in twenty-four to thirty-six hours. However, clinical relapse of the pharyngitis-tonsillitis occurred in 4 of the 9 patients treated for less than four days but in none of the 10 treated for six days. One patient treated with penicillin for six days subsequently developed acute hemolytic streptococcic sinusitis and pneumonia. Throughout the study relapses and complications always were associated with the presence or reappearance of hemolytic streptococci in the nasopharynx.

5. This study reveals further evidence of the high antihemolytic streptococcic action of penicillin and emphasizes its value in dangerous hemolytic streptococcic infections. Penicillin in minor hemolytic streptococcic infections may be beneficial, but further investigation will be required to ascertain its exact indications and contraindications in these conditions.

Cone [12] used a prophylactic local infiltration of penicillin just prior to tonsillectomy in 21 cases. All of the patients were observed for reactions. The solution contained 5,000 Oxford units of penicillin to 1 cc. of isotonic solution of sodium chloride. From 1 to 2 cc. of the solution was used in a deep peritonsillar injection immediately after the routine infiltration with a 1 per cent solution of procaine hydrochloride. In a few cases the penicillin was mixed with 10 cc. of a 1 per cent solution of procaine hydrochloride. In no case was there an untoward reaction. From a clinical standpoint healing was facilitated. Four cases of severe peritonsillar or parapharyngeal infections are reported in some detail.

Joseph [13] calls attention to the disadvantages and unsatisfactory results of older methods of treatment of Vincent's infection of the tonsils and reports a study of the efficacy of penicillin as used in the treatment of this infection. Most of the patients selected for the study had each a necrotic, ragged tonsillar lesion; gingival lesions were not included. Smears were taken routinely, but the final diagnosis was based on the history of the case and the appearance of the lesion. During the first part of the study the mode of administration and the size of dose were

12. Cone, A. J.: Local Infiltration of Penicillin Solution in Peritonsillar and Pharyngomaxillary Spaces, Ann. Otol., Rhin. & Laryng. **54**:84-89 (March) 1945.

13. Joseph, G. F.: Penicillin Therapy for Vincent's Ulcers of the Tonsils, South. M. J. **38**:778-781 (Dec.) 1945.

varied in order to arrive quickly at the most practical method and the smallest effective dose. Fifty-one patients were treated; 10 received 50,000 units, 13 received 25,000 units and 3 received 12,500 units. Two patients were treated by topical application, and 5 were given the drug orally in cottonseed oil.

The author's summary follows:

(1) A controlled study to evaluate penicillin in the treatment of Vincent's tonsillitis is described.

(2) It was found that 100,000 units of penicillin in two 50,000 unit intramuscular doses three hours apart provide optimal treatment for Vincent's ulcers of the tonsil.

Twining, Szylejko and Kern [14] report their success in treating 41 patients suffering from Vincent's infection with penicillin. The diagnosis was based on the clinical and laboratory findings. Twenty-nine of the patients were cured by one course of ten intramuscular injections, 20,000 units of penicillin being given every three hours. The remainder were cured by a second similar course given the following day. The patients were considered cured when they were free from all symptoms, the lesions were healed and there was a negative smear for Vincent's organisms. The average number of days spent in the hospital was seven for the penicillin-treated patients, compared with twenty-three days for those treated by other methods.

MacGregor and Long[15] report the results of an experiment carried out to determine the amount of penicillin that would reach the affected areas in the back of the mouth when the drug was administered in the form of gelatin pastilles. In the experiment gelatin pastilles containing 1 per cent methylene blue were placed in the buccal fold between the cheek and the teeth and allowed to dissolve without agitation. The mouths were examined at intervals of five and fifteen minutes after insertion. Four areas were noted: the anterior faucial pillars, the uvula, the tonsil or tonsillar fossa and the pharynx. At the end of five minutes there was good staining of all areas except the pharynx; at the end of fifteen minutes the staining of all areas had increased but staining of the pharynx continued slight. As a result of their observations the authors conclude that the posterior wall of the pharynx cannot be reached with drugs in effective concentration from the mouth and that it thus differs from the tonsillar area.

Robinson [16] reports a study of hemolytic streptococcus infection of the throat and its complications based on 77 cases seen during the latter

14. Twining, H. E.; Szylejko, H. W., and Kern, R. A.: Penicillin in the Treatment of Vincent's Angina, U. S. Nav. M. Bull. **45**:479-481 (Sept.) 1945.

15. MacGregor, A. B., and Long, D. A.: Local Spread of Drugs in the Oral Cavity, Lancet **2**:299-300 (Sept. 8) 1945.

16. Robinson, P.: Haemolytic Streptococcal Throat Infections: Their Complications and Sequels, with Special Reference to Penicillin Treatment, Brit. M. J. **2**:213-214 (Aug. 18) 1945.

part of 1944 and the early part of 1945. The cases are divided into two groups. The first group covers the period from November 1944 through January 1945; in this group there were 34 cases of tonsillitis, in 2 of which peritonsillar abscess was present, 3 cases of pneumonia, 1 case of scarlet fever, 4 cases of erythema nodosum and 6 cases of acute rheumatism. The second group of cases includes those observed in February, March and April 1945; it consisted of 43 cases of tonsillitis, including 9 in which peritonsillar abscess was present, 7 cases of erythema nodosum and 11 cases of acute rheumatism.

Attempts to sterilize the throats in the first group by frequent spraying with sulfathiazole powder or calcium penicillin powder failed completely. Sulfathiazole was then given by mouth, 20 to 30 Gm., but with no better results; similar treatment, in group 1, of peritonsillar abscess, erythema nodosum and acute rheumatism was not satisfactory.

The second group of patients was treated with penicillin pastilles prepared according to the technic of MacGregor and Long. Ten pastilles were given daily at one and one-half hour intervals; the number used per patient varied from 18 to 46. Cultures were made on admission to the hospital and on completion of the treatment. No recurrence of symptoms and no complications occurred in this group.

The author summarizes the article as follows:

Acute haemolytic streptococcal tonsillitis and peritonsillar abscess respond well to treatment with penicillin pastilles.

Erythema nodosum following haemolytic streptococcal tonsillitis may be aborted, and possibly prevented, by penicillin pastilles.

Some cases of rheumatic fever seem to respond to systemic penicillin treatment.

Rosenthal [17] gives two formulas for lozenges to be used in the local treatment of severely infected throats; one contains penicillin and the other a sulfonamide compound. Remarkable results are claimed for penicillin lozenges in cases of follicular tonsillitis and ulcerous Vincent's infection of the throat. The medication goes into solution in approximately fifteen minutes. One lozenge is given hourly. After administration, according to the observations of the author:

(1) There is dramatic relief of pain on deglutition within 4 hours.

(2) Tonsillar edema and necrotic tissue are reduced as much as 25 to 50 per cent within 12 hours.

(3) Toxic appearance and fever usually vanish within 24 to 36 hours.

Sulfonamide Compounds.—Clodfelter [18] reports a study of the results of treatment with sulfonamide compounds carried out in 400 selected

17. Rosenthal, H. H.: Simple Local Treatment for Throat Infections, U. S. Nav. M. Bull. **45**:75-76 (July) 1945.

18. Clodfelter, H. M.: The Treatment of Severe Acute Tonsillitis With and Without Sulfonamides, Ohio State M. J. **41**:819-820 (Sept.) 1945.

cases of acute infection of the upper respiratory tract of such severity as to require hospitalization. There was a control group of 168 cases in which no patient received sulfonamide compounds.

On admission to the hospital the average temperature of the 400 patients was 102.8 F. The average of the highest temperatures reached by the control group was 103.4 F. The average of the highest temperatures reached by the treated group was not above the temperature on admission. In the control group the average temperature rose during the first forty-eight hours after admission to 103.8 F.; the temperature dropped to normal or below on the fourth day, and the patients were discharged after the temperature remained normal for twenty-four hours, the average period spent in the hospital being 5.3 days. In the treated group there was a drop in the temperature during the first twenty-four hours; the normal level was approached on the third day, but on the fifth to the seventh day there was a rise followed by a slow drop to normal on the ninth day. These patients were kept in the hospital until the temperature was normal for twenty-four hours for an average of 9.7 days; on discharge from the hospital these patients did not look well, did not feel well and were unwilling to return to duty. In his concluding sentence the author states that "it seems highly suggestive that the use of sulfonamides in the treatment of acute 'strep' tonsillitis is not only of no benefit but actually detrimental to the welfare of the patient."

Wiesel and Teague [19] report the results of a study started in 1941 in which a 5 per cent solution of sulfathiazole sodium sesquihydrate was used as a gargle in the treatment of acute pharyngitis and tonsillitis. Over 200 patients were treated in this manner; no controls were used. The first 100 patients were almost all adults with mild or moderately severe pharyngitis and with a temperature less than 100 F. The results were satisfactory, and recovery seemed more rapid than was expected with the use of conventional methods of treatment. The second 100 patients included those with acute tonsillitis as well as those with pharyngitis. Rapid reduction of swelling of the tonsil and disappearance of the exudate when present were noted. One patient experienced a sudden reaction of fever, rash, edema and arthralgia after using the gargle for four days, which was interpreted as a drug reaction. After a course of treatment in which 30 cc. of the gargle was used every two hours, there was a slight but progressive rise of the blood sulfathiazole level to an average of 0.9 mg. per hundred cubic centimeters of blood in seven hours.

19. Wiesel, B. H., and Teague, R. S.: Acute Pharyngitis and Tonsillitis. Treatment with Sodium Sulfathiazole Gargle, with Experimental Studies on the Extent of Absorption, J. M. A. Alabama **14**:233-236 (April) 1945.

Bismuth Compounds.—Silber [20] calls attention to a previous communication reporting the effective use of theobroma oil suppositories containing the bismuth salt of heptadienecarboxylic acid in a series of cases which included tonsillitis (follicular, catarrhal and parenchymatous), nasopharyngitis and gingivostomatitis. The results indicated such advantages as speedier recession of symptoms, which disappeared usually within forty-eight hours, more rapid loss of fever, rapid disappearance of the objective findings, greater ease of administration and complete absence of either local or systemic toxic effects.

According to the author, there are still several unsettled matters which the present article is intended to clarify further:

. . . (1) the urinary picture before, during, and after treatment; (2) the presence or absence of the specific evidences of bismuth toxicity, namely, pigmentation, stomatitis, jaundice, nephritis, dermatitis, or other notable reactions during or after treatment; (3) the presence or absence of evidences of local injury about the site of administration, rectum, anus, lower bowel; (4) the absorption of bismuth as measured by urinary excretion; (5) the margin of safety as measured by the lack of toxicity from the use of more than two suppositories; (6) the incidence and distribution of organisms encountered at the site of the infection; (7) the effectiveness of the treatment as measured against the results obtained by the use of alternate patients treated by other methods as controls.

The article is well summarized by the author as follows:

1. Twenty-one patients with tonsillitis pharyngitis with varying degrees of of adenitis, and gingivostomatitis were treated with Analbis suppositories containing 0.0225 Gm. (children size) and 0.045 Gm. (adult size) metallic bismuth each, in the form of heptadienecarboxylic acid salt of bismuth.

2. Five of these had unrelated conditions: two had scarlet fever; one, rheumatic fever; one, adenitis following scarlet fever and measles; and one had sepsis. These cases were excluded from certain averages, in this study, that were not pertinent.

3. Twenty alternate patients were treated as controls, seventeen of the twenty symptomatically, the remaining three with sulfonamides.

4. There were no evidences of toxic bismuth effects, either local or systemic, irrespective of the number of suppositories given.

5. There were no evidences of clinical nephritis, nor of any damage of the kidneys on urinalysis in any of the patients treated with Analbis.

6. The mean urinary excretion of bismuth in thirteen patients followed over a period of four days after first administration of Analbis was 2.180 mg. per day per patient. Total urinary excretion in the same thirteen patients over a period of four days amounted to 10.09 per cent of the total amount of bismuth administered.

7. The organisms encountered in stomal infections were cocci in forty cases; staphylococci as a group in thirty-eight cases; albus thirty-five cases, aureus, thirty-three cases, and citreus, one case; streptococci as a group in twenty-five cases, hemolyticus, sixteen cases, viridans, twelve cases and non-hemolyticus, two cases; micrococcus catarrhalis in twenty-three cases; Vincent's bacillus in two cases; pneumococcus type XIII in one case.

20. Silber, S.: Bismuth Suppositories (Analbis) in Throat Infections, J. Pediat. **25**:245-253 (Sept.) 1944.

8. Treatment with Analbis was more effective than symptomatic treatment.

9. Recovery was effected in cases of tonsillitis and pharyngitis in 2.68 days in fourteen patients treated with Analbis as against 6.3 days in fifteen patients treated symptomatically and 5.3 days in three cases treated with sulfonamides.

10. Recession of temperature to normal in the fourteen patients treated with Analbis occurred in 2.18 days, in fifteen patients treated symptomatically in 5.87 days, and in three patients treated with sulfonamides in 5.3 days.

11. In four patients with gingivostomatitis treated in this series Analbis medication showed advantages over other forms of treatment comparable to those obtained in tonsillitis and pharyngitis.

12. The average number of Analbis suppositories used in twenty cases was 2.70.

Stovin [21] has evolved a routine in the preoperative and postoperative care of patients undergoing tonsillectomy which is designed to restore them to their normal state with the least amount of pain and discomfort in the shortest possible time. Before the operation the patient is given a diet high in sugar content. A preoperative laxative is rarely given. The author believes that localized pain, general malaise and delayed healing of the wound are due to secondary infection. To reduce these symptoms, to hasten healing and to shorten the period of convalescence he gives 2 "analbis" suppositories (theobroma oil containing the bismuth salt of heptadienecarboxylic acid) in dosage according to age; the first is given twenty-four hours before the operation, and the second immediately before it. After the operation reenforced feedings are given as soon as possible, usually within four hours. The feeding consists of 8 ounces (240 cc.) of milk with the addition of 1 ounce (30 cc.) of cocomalt, and in most cases this feeding is repeated every few hours. On the following day, semisolid foods are given, and the patient is encouraged to return to a normal diet as soon as possible; he is also encouraged to chew gum between feedings. The author states that the results following the routine described have been impressive. In almost every case he observed reduction of the duration and the intensity of the postoperative pain and lessening or absence of the malaise. He believes these results are due to the high caloric feedings immediately before and after the operation and to the bactericidal properties of the bismuth.

POSTOPERATIVE HEMORRHAGE

Neivert [22] reports a study of the cause and the prevention of late tonsillar hemorrhage. He states that certain components of the blood which may lead to a solution of the problem have been studied, and he directs attention to the works and observations of others which suggest that the preoperative and postoperative administration of acetyl-

21. Stovin, J. S.: Preoperative and Post-Operative Care in Tonsillectomy, Eye, Ear, Nose & Throat Monthly **24**:239-242 (May) 1945.

22. Neivert, H.: Late Secondary Tonsillar Hemorrhage: Studies of Prothrombin and Vitamin K, Arch. Otolaryng. **42**:14-18 (July) 1945.

salicylic acid is a prominent factor in these hemorrhages. The author and his associates determined the prothrombin time in a large number of cases; they consider a prothrombin time of seventeen to twenty-four seconds normal, according to the technic of Page. They found that a total daily dose of 2.4 Gm. of acetylsalicylic acid resulted in an increase-K-of the prothrombin time, sometimes within twenty-four hours. Further study revealed that the administration of the water-soluble vitamin like compound "synkayvite" (tetrasodium 2-methyl-1, 4-naphthohydroquinone diphosphoric acid ester) prevents the increase in the prothrombin time. In one paragraph the author states that

> On the basis of these laboratory findings we decided to evaluate the combined acetylsalicylic acid-"synkayvite" medication clinically on a broader scale. We employed both the rectal suppositories (formula already given) and the tablets composed of 0.3 Gm. (5 grains) of acetylsalicylic acid and 5 mg. of "synkayvite." Two of these tablets, it was believed, would give a satisfactory analgesic effect and at the same time afford full protection against the prothrombinopenia induced with acetylsalicylic acid. Furthermore, it appeared that such combined oral medication would have an advantage over the administration of acetylsalicylic acid and "synkayvite" in individual tablets. Thus far we have given the suppositories to 173 [patients] and the combined tablets to 110 patients. Results were most gratifying, with only 4 patients troubled by late tonsillar bleeding among these 283 patients, an incidence of 1.4 per cent. Incidentally, one of the late hemorrhages encountered in this series occurred in a 6 year old child in whom the bleeding undoubtedly was due to thumb sucking. She always created a powerful suction action as she withdrew the thumb from her mouth.

The author summarizes his article as follows:

> While in rare cases the cause of late post-tonsillectomy hemorrhage may be trauma, menstruation, infection, deficiency of vitamin C or some blood dyscrasia, our observations to date suggest that one of the most important factors is a reduction in the prothrombin of the blood brought about by the use of acetylsalicylic acid or salicylates. The hypoprothrombinemia interferes with coagulation. However, with the simultaneous administration of a vitamin K-like compound ("synkayvite") the prothrombin-lowering effect of acetylsalicylic acid will be overcome and consequently normal coagulation can take place.

Singer [23] in discussing post-tonsillectomy hemorrhage states that following tonsillectomy two types of postoperative course may be observed, one described as normal, in which hemorrhage does not occur, the other described as abnormal, in which there is a characteristic hemorrhagic inflammation of the soft palate and which is often complicated by secondary hemorrhage. If the healing process is abnormal, on the third day a nonpainful hemorrhagic suffusion of the margins of the wound develops and quickly spreads into the soft palate and the uvula; when this occurs the membranes covering the fossa have a dirty gray appearance. Frequently, in these cases, on the sixth

23. Singer, R.: Acetylsalicylic Acid: A Probable Cause for Secondary Post-Tonsillectomy Hemorrhage, Arch. Otolaryng. **42**:19-20 (July) 1945.

or seventh postoperative day, following an attack of radiating pains, a secondary hemorrhage occurs; the bleeding is usually unilateral, but it may be bilateral, and is sometimes recurrent.

The author states that the incidence of post-tonsillectomy hemorrhage is negligible in the operating clinics of central Europe, where acetylsalicylic acid is not used in the preoperative and postoperative regimens. He reports a series of 75 cases observed during 1943 in which acetylsalicylic acid was not used, and there was no postoperative hemorrhage and no hemorrhagic inflammation of the soft palate. The author states that "the prothrombin-lowering effect of salicylates described by a number of authors seems to be a plausible explanation for the occurrence of secondary post-tonsillectomy hemorrhage."

Campbell [24a] in 1943 published an article on the subject of bleeding after tonsillectomy and adenoidectomy; in a group of 225 cases there was postoperative oozing in 2 cases; both patients had received an inadequate amount of menadione. Previous to the use of this analog of vitamin K oozing occurred in more than 3 per cent of the author's cases.

His present study [24b] is based on 198 unselected cases in which menadione and, later during the study, ascorbic acid were used. These substances were combined in capsules and their administration was started four days before operation; it was continued for eleven days after operation but was omitted on the day of operation. The author reports that postoperative bleeding occurred in 3 of this series of 198 cases.

ANESTHESIA

Fish,[25] discussing anesthesia in connection with tonsillectomy, states that from the standpoint of the anesthetist there is no other minor operation which requires more skill in technic and better judgment than the supposedly simple tonsillectomy and adenoidectomy. To insure a safer and more satisfactory induction of anesthesia in these cases, he has devised a nasotracheal procedure of intubation and administration of "fractional gas-ether," which he describes as follows:

> The nasal approach is used with a Magill tube of sufficient size to pass readily through the nasal passages, to provide adequate airway, and at the same time to block off the laryngeal entrance to the trachea. The tube may be displaced to right or left by the suction tip as it becomes necessary. The tube will thus serve as a barrier to keep the suction tip out of the tonsil fossa on the operated side, and consequently avoid aspiration of clot from the cut ends of blood vessels.

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24. Campbell, C. A.: (*a*) Vitamin K in Tonsillectomy Granulation Oozing, Ohio State M. J. **39**:1116 (Dec.) 1943; (*b*) The Second Report of Prophylaxis of Post-Tonsillectomy Granulation Oozing, ibid. **41**:621-622 (July) 1945.

25. Fish, F. H.: Specialized Anesthesia for Tonsillectomy, Anesth. & Analg. **24**:259-260 (Nov.-Dec.) 1945.

He claims for the method the following advantages: (1) The duration of the operation is not prolonged, (2) the amount of the anesthetic agent used is less, (3) the level of the anesthesia is better controlled and (4) there is less risk of pneumonia and pulmonary abscess resulting from aspiration of detritus during the operation.

Nichols [26] briefly discusses some of the difficulties encountered in the induction of general anesthesia for tonsillectomy and states that the main difficulty arises from the necessity for the anesthetic agent to pass through the field of operation. To avoid some of the difficulties, he attempts to have the patient brought to the operating room under the influence of a suitable sedative and with stomach empty. Anesthesia is induced with ethyl chloride and ether. As soon as the patient's jaws relax, a Magill tube is introduced through the mouth into the larynx with the aid of a laryngoscope. A mouth gag is immediately dropped over the oral end of the tube. The Magill tube is connected to an ordinary 1 pound (0.5 Kg.) can of ether with a tube attached to the central opening and with several other openings punched in the top of the can; by opening and closing these holes the concentration of vapor is regulated to the requirements of the patient.

Advantages claimed for this method are a smoother induction of anesthesia, better aeration and greater ease and safety in the control of hemorrhage.

Griffiths and James,[27] after using pentothal sodium in the induction of general anesthesia for tonsillectomy in 133 cases, recommended it as a safe reliable anesthetic for this operation. In their experience it has proved eminently satisfactory in regular hospitals and in wholly tented hospitals. Its administration requires considerable experience of pentothal anesthesia on the part of both the anesthetist and the surgeon; it is not recommended for only occasional use. Its safety and success depend on the maintenance of a perfectly clear airway at all times and the avoidance of any reflex laryngeal stimulation until the patient is sufficiently deeply anesthetized.

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26. Nichols, T. R.: Inhalation Anesthesia for Tonsillectomy, Canad. M. A. J. **52**:378-379 (Oct.) 1945.

27. Griffiths, H. F., and James, J. A.: The Use of Pentothal for Tonsillectomy in Adults, J. Laryng. & Otol. **60**:28-30 (Jan.) 1945.

Abstracts from Current Literature

Ear

Otic Complications of Streptomycin Therapy. Edmund P. Fowler Jr., and Ewing Seligman, J. A. M. A. **133**:87 (Jan. 11) 1947.

The authors discuss symptoms referable to toxic involvement of the vestibular and auditory systems following streptomycin therapy. They give several case reports including tables, charts and graphs. They summarize their findings and conclusions as follows: 1. Eighty-one streptomycin-treated patients were examined for evidence of aural toxicity. 2. Two months after the cessation of therapy 3 of these patients showed an absence of vestibular response. During the third month after therapy 1 of these 3 showed increasing recovery of response. All 3 complained spontaneously of dizziness during treatment. This symptom has decreased steadily and is at present not incapacitative. Except for 1 patient who had a probably unrelated high tone loss, these 3 patients had normal audiograms. 3. Another patient showed a severe loss of hearing two weeks after treatment. One month after treatment he showed decided improvement in hearing. He had a normal vestibular response throughout. 4. Because of the possibility of complications, the indications for the use of streptomycin should be carefully considered in each case. However, when indicated, the drug should not be withheld because of this danger. If otic symptoms occur during streptomycin therapy, the case should be reconsidered before the administration of the drug is continued. If the therapy is continued, streptomycin with another manufacturer's lot number should be tried. 5. An audiogram and a vestibular test should be performed before the initiation of streptomycin therapy. 6. The low tone and high tone losses here reported are probably not due to streptomycin except in 2 cases (1 and 5). The low tone losses are probably due to outside interference. The losses in high-pitched tones, relatively unimportant in the hearing of speech, are the type commonly found in soldiers who have served in active combat.

Gordon, Philadelphia.

Sound Location and Its Determination in Connection with Some Cases of Severely Impaired Function of the Vestibular Labyrinth, but with Normal Hearing. Herman Diamant, Acta oto-laryng. **34**:576 (Nov.-Dec.) 1946.

Cases in which, despite normal hearing, the function of the vestibular nerve is impaired are of interest from the standpoint of the location of sound. There are two theories on the locating of sound. According to the first the capacity to do this depends on the sound itself, particularly on its intensity and tone color and also on the difference in time between the two aural perceptions of the sound. According to the other theory, the decisive role is played by the sense of equilibrium. "By the unconscious movements made in trying to locate the origin of a sound, impulses are transmitted centrally via the semicircular canals." The first theory is generally accepted, but some authors, notably Güttich and Roth, believe that the vestibular labyrinth assists in the location of sound.

Diamant examined 3 patients with normal hearing but seriously impaired vestibular function. In 1 the vestibular function was completely abolished, and in 2 only insignificant traces remained. The testing apparatus consisted of a tone generator with an amplifier and two loud speakers suspended at a distance of 6.5 meters from the ear to be tested, and movable from side to side. A tone was produced and transmitted by the loud speakers, first one and then the other, quite

at random. The pitch of the tone varied for the different tests, which numbered at least ten for each distance. The subject was required to judge from which of the loud speakers the sound emanated. The loud speakers were moved nearer to each other until they were separated by a distance at which it was no longer possible to judge the position of the source of the sound, whether it was from the right side or from the left. The angle between lines of an isosceles triangle between the subject tested and the two loud speakers could then be computed. Besides the 3 subjects just mentioned, 15 persons whose ages ranged between 21 and 50 years and whose hearing and vestibular reactions were normal were also tested.

In the 15 normal persons the angle which Diamant calls the "sound location angle" varied between 6.6 and 16.6 degrees, with an average of 11.7 degrees. With his first pathologic subject, with a total absence of vestibular function, the "sound location angle" was 15.8 degrees, this being well within the limits of normal deviation; with each of the other 2, with seriously impaired vestibular function, the "sound location angle" was 11.4 degrees.

These tests were all made on blindfolded patients, who were, however, permitted to move their heads at will. From these tests Diamant concluded that the vestibular function does not play as great a role in the location of sources of sounds as Güttich and Roth have maintained.

GROVE, Milwaukee.

Pharynx

ANOMALIES IN THE COURSE OF THE INTERNAL CAROTID ARTERY IN THE UPPER REGION OF THE PHARYNX. B. BERGQUIST, Acta oto-laryng. **34**:246, 1946.

Carmack came across 4 cases of tonsillectomy in two years in Indiana in which death followed hemorrhage. In 2 of the cases the hemorrhage was a fulminating one, occurring at the time of the operation, and in the other 2 the death from hemorrhage occurred later. Conolly in 1913 described a case in which a loop formed by the internal carotid artery across the upper pharyngeal wall was cut off on one side.

Bergquist reports the following case: A child of 5 underwent an operation for removal of tonsils and adenoids. After the median adenoid mass had been curetted and then the lateral, the area was cleaned out by suction. After this there could be seen on the posterior pharyngeal wall a pulsating vessel as thick as a pencil. It had not been injured in the curettage of the adenoid vegetations. It was so large that it filled the space between the posterior pillar and the midline and had the appearance of the carotid artery. It entered the lateral pharyngeal wall some centimeters above the introitus laryngis, proceeded upward near the midline to the base of the skull, where it turned laterally toward the carotid canal in the apex of the petrous bone. The throat healed well, and after healing had taken place the abnormal vessel could not be seen but its pulsations could still be palpated.

Bergquist states that ordinarily the internal carotid artery exhibits a rather straight course from the bifurcation at the level of the upper edge of the thyroid cartilage to its entrance into the carotid canal and is usually placed at a distance of 2 to 2.5 cm. from the lateral pharyngeal wall. In his case it made a wide loop medially to the pharyngeal wall and between the mucous membrane and the muscle support where it lay concealed by a well developed mass of adenoids. He states that abnormalities of the internal carotid artery are not unusual in this region. In 1925 Kelly collected from the literature 150 cases of abnormality of the vessels of the upper region of the throat. In 85 of these the abnormal vessel could be seen and in 65 the pulsations could be palpated. Not all of the abnormalities were due to aberrations of the position of the internal carotid artery. Some of them were probably abnormalities of the ascending pharyngeal artery, but Kelly concluded that the pulsations on the posterior pharyngeal wall were probably those of the internal carotid artery.

Another differential diagnosis to be considered in connection with pulsations in the throat concerns aneurysms, but Bergquist considers these to be relatively rare.

Bergquist states that in cases in which vessels are injured during the operation for removal of tonsils and adenoids the patient usually reaches the postmortem table, as the fulminating hemorrhage occurring is hard to check.

GROVE, Milwaukee.

Larynx

ETIOLOGIC FACTORS OF CENTRAL PARESIS OF THE RECURRENT NERVE. NILS O. KRUSE, Acta oto-laryng. **34**:554 (Nov.-Dec.) 1946.

An etiologic classification of cases of paresis of the recurrent nerve divides them into (1) cases of the peripheral and (2) cases of the central type. Cases of the central type can be further subdivided into (*a*) cases of supranuclear and (*b*) cases of nuclear or bulbar type. Kruse directs attention to the fact that peripheral paresis of the nerve is by far the more common type and states that many factors may affect each recurrent nerve during its long and tortuous course from the point at which it makes a common exit from the medulla with the tenth nerve to its ultimate distribution. He also stresses the point that in a case of acute paresis of the nerve the exact etiologic factor is frequently not found in spite of the most exact clinical, roentgenologic, serologic and endoscopic examinations. In such a case a diagnosis of paresis of the recurrent nerve of unknown cause is put down. This diagnosis frequently must be altered, on later examinations, to paresis due to esophageal carcinoma or to mediastinal tumor or to some other factor not present at the first examination.

Among the published series of cases of paresis of the recurrent nerve are those of Smith, Lambert and Wallace, and New and Childrey; there are listed in all 452 cases, 27, or 6.9 per cent, of which were listed as instances of paresis of central origin. In a recent series of 141 cases of Frenkner 13.5 per cent were listed as instances of paresis of central origin. Kruse thinks that both of these percentages are too high, because in the series of New and Childrey there were 9 and in the series of Frenkner 11 in which the patients were tabetic. These cases, he thinks, should have been classified as instances of paresis of peripheral origin.

Of the cases of central paresis, those of the supranuclear type are rare, and the paresis is due to such lesions as abscess of the brain, hemorrhage, thrombosis, embolism, neoplasm, solitary tubercle and syphilitic involvement of the cerebral cortex and the brain stem. That laryngeal paralysis is so seldom seen in association with these lesions is due to the course of the corticobulbar tracts, which, according to Furstenberg, run from their place of origin in the precentral gyrus through the internal capsule, the cerebral peduncles and the pons, partly enclosed in the pyramidal tract, and end in the medulla after partial decussation. The motor vagus nucleus is located in the nucleus ambiguus. The fact that the corticobulbar tracts pertaining to the larynx are both decussated and nondecussated explains why paresis of the larynx of the supranuclear type is so rare. For a cortical lesion to produce a clinically ascertainable paresis of the larynx it must be bilateral and symmetric or, as Furstenberg has pointed out, it must extend to at least four fifths of the pons or the cerebral peduncles.

The cases of nuclear paresis of the larynx constitute the main group of cases of central paresis and can be divided into those of acute and those of chronic form. The acute form is caused by embolism, hemorrhage, thrombosis and acute encephalitic involvement of the bulbar portion of the brain. The chronic form, the cases of which are more numerous than those of the acute form, occurs during the course of a chronic disease of the central nervous system, such as syringomyelia, amyotrophic lateral sclerosis, disseminated sclerosis, progressive bulbar paralysis, syphilis of the brain, neoplastic growth, etc. The terms "acute" and "chronic"

do not refer to the mode of onset of the laryngeal paresis but rather to the etiologic character of the causal agent. The paresis may come on as suddenly in one group as in the other.

Kruse reports 39 cases of paresis of the recurrent laryngeal nerve studied in his hospital in a six year period. These are the basis of his report. He also mentions 81 cases seen in the outpatient department during this same six year period; in the majority of these the condition was due to a preceding thyroidectomy. These 81 cases are not included, however, in his report. Of the 39 cases, 2 were instances of the nuclear and the rest of the peripheral type. This, in a small series, gives the percentage of cases of paresis of central origin as 5.1, which agrees fairly well with the figures for the other published series mentioned.

Kruse reports 2 cases in detail:

CASE 1.—A woman aged 26 was taken ill with an acute infection of the upper respiratory tract, and on the following day paresis of the left recurrent laryngeal nerve developed. Increasing dyspnea soon required that tracheotomy be performed, but in spite of this the patient rapidly succumbed. At the autopsy the significant findings concerned the brain and more particularly the brain stem. Microscopic examination of this revealed marked encephalitis in the medulla and the pons, especially around the fourth ventricle. This encephalitic process, though diffusely spread throughout the medulla, was most marked in an area beneath the floor of the fourth ventricle, extending ventrally up to the nuclei of the vestibular nerve and the fifth cranial nerve. There was nothing in the complete autopsy to indicate any peripheral involvement of the left recurrent nerve.

CASE 2.—In a woman aged 62 paralysis of the left recurrent nerve had developed following a thyroidectomy performed twenty years earlier. For the last four or five years she had suffered from attacks of jacksonian epilepsy involving principally the right arm in association with temporary aphasia. Arteriography was carried out, a colloidal suspension of thorium dioxide being used as the contrast medium. Eleven days thereafter the patient suddenly became hoarse, and considerable dyspnea developed. Examination of the larynx revealed that to the old post-operative paresis of the left recurrent nerve there had been added acute paresis of the right vocal cord. After three to four days the stridor had disappeared, and on discharge twelve days later the paresis of the right vocal cord had completely abated.

In this case, Kruse believes, the paresis of the right vocal cord, occurring eleven days after the arteriographic use of a contrast medium, was caused by thorium dioxide obstruction of a small vessel of the nucleus ambiguus.

Among the various reported series there are a number of cases of paresis of the recurrent laryngeal nerve of unknown cause. The diagnosis of "rheumatoid neuritis" must always be considered open to question, especially if repeated examinations several years later still reveal the paresis, even though, at this time, no known etiologic factor for the paresis has been discovered. Kruse quotes Gerber as of the opinion that this diagnosis is justifiable only if it can be shown that the paresis has disappeared without sequelae.

Kruse cites 3 cases of paresis of the recurrent nerve with a physical examination giving entirely negative results except for an increase of the protein content of the spinal fluid unassociated with any increase of the cell count. He offers the hypothesis that in these cases the paresis may have been due to transitory acute encephalitis of the bulbar part of the brain. That acute encephalitis of the medulla does not always spread along a wide front but seems to concentrate in certain areas and to spare others is evidenced by a case reported by Fremel and also by a fatal case encountered in his own clinic in which the only symptom of involvement of a cranial nerve was unilateral paresis of the recurrent nerve.

GROVE, Milwaukee.

Nose

NASOPHARYNGEAL ATRESIA WITH REPORT OF A NEW PROCEDURE BY SKIN GRAFT TO THE LATERAL PHARYNGEAL WALL. H. S. VAUGHAN, Plastic & Reconstruction Surg. **1**:309 (Nov.) 1946.

Two types of atresia are considered by Vaughan: true atresia, which is congenital and the result of an arrest of development; secondary atresia, which may be due to adhesions formed between the soft palate and the pharyngeal wall. The former is the expression of a failure of the embryonic foregut to open, whereby the pharynx continues to be separated from the mouth and the nasal cavity. The symptom of nasal atresia results from interference with nasal breathing, and may even be complete stoppage, although so extreme a degree is relatively rare. Lack of an adequate passageway for secretions leads to infection of the nasal cavity and of the eustachian tubes, with consequent deafness. When the condition has been acquired, ease of reconstruction depends on the degree of mucosal damage and the amount of scar tissue which has formed. The results of operation are usually more successful if the condition has followed simple trauma or an inflammation than if it is due to asyphilitic lesions. The object of operations, regardless of the scar contraction which is present, is to keep the space open. The McKenty method is recommended when there is only little scar tissue, but the author has found it less satisfactory if there is dense overgrowth of scar tissue. In the method presented, a silver diaphragm is used to keep the denuded surfaces separated.

SELTZER, Philadelphia.

THE REPAIR OF NASAL LOSSES. F. YOUNG, Surgery **20**:670 (Nov.) 1946.

Young describes the functions of the nose and gives in considerable detail methods for the correction of various nasal defects and deformities. Each is adapted to the individual case, and depends on the type and the degree of deformity. The article is well illustrated. It is too diverse to permit a brief abstract.

SELTZER, Philadelphia.

SURGICAL CORRECTION OF NASAL DEFORMITIES. L. T. BYARS, Surg., Gynec. & Obst. **84**:761 (Dec.) 1946.

Byars gives in some detail methods for the correction of (1) large hump nose, (2) hanging columella, (3) cleft lip, (4) deviated septum, (5) snubbed nasal tip, (6) nasal asymmetry with harelip, (7) traumatic septal deformity, (8) depressed (saddle) nose and (9) rhinophyma. The article is well illustrated, but presents no new method.

SELTZER, Philadelphia.

Miscellaneous

PROSTHETIC RESTORATION OF CONGENITAL CLEFT PALATE. N. A. OLINGER, Am. J. Orthodontics **32**:456 (July) 1946.

Olinger reviews the different types of cleft palate and their treatment. Stress is laid on the desirability of the decision for procedure being made jointly by the surgeon and the dentist. In his opinion, there is a lack of general realization of the large number that can be helped. Two main factors are considered: (1) improvement of tonal quality and of speech; (2) maintenance of a healthy condition of oral and nasal passages. If the vomer is absent, an artificial one is constructed, since the vomer has a specific physiologic function. Its secretions and the fluid that condenses on it prevent the pharyngeal walls from becoming incrusted with mucus, with consequent development of bad breath and digestive disturbance. In the absence of the soft palate, an artificial velum helps in the formation of sound. For successful treatment there should be (1) a careful roentgen examination and (2) an impression made with any suitable material. A velum is attached to the denture by a hinge, which allows it to hang naturally

in the nasopharynx. The musculature of the pharynx and the palate are strengthened by the repetition of suitable sounds so that they will close firmly around the artificial velum and prevent sound from reaching the nasal cavity. The improvement of speech and articulation depends to a large degree on securing this condition. Speech lessons should follow the installation of the prosthesis.

SELTZER, Philadelphia.

RECONSTRUCTIVE OTOPLASTY. P. W. GREBLEY, Arch. Surg. **53**:24 (July) 1946.

Greeley discusses the observation that fresh isografts of auricular cartilage are apt to undergo absorption, in contrast to transplants of conchal cartilage and isografts of costal cartilage. A single case is described, and the result of the use of a fine mesh tantalum wire model is compared with the results obtained with several other materials. The wire mesh has the advantage of lightness, and it also allows penetration of a vascular blood supply. In his single case the result was satisfactory. The wire mold was embedded under a delayed temporomastoid skin flap, where it was allowed to remain untouched for seven months. The structure was then dissected up and brought forward, and the posterior surface and the area of the former bed were covered with stent graft. Complete healing took place in three months. Two small areas of pressure necrosis developed on the peripheral margin, apparently from contracture of scar tissue in the skin around the periphery of the mold. The scars were corrected by the use of a small tube pedicle flap from the neck (Pierce). After a period of twenty-five months the condition was satisfactory.

SELTZER, Philadelphia.

REPORT OF A DEATH FROM PONTOCAINE HYDROCHLORIDE. WILLIAM A. AHROON, Laryngoscope **56**:320 (June) 1946.

Ahroon presents a case of death resulting from local applications of a solution of "pontocaine hydrochloride" (tetracaine hydrochloride U. S. P.). The solution was sprayed into the nose, the pharynx and the trachea. The patient had gone through a similar procedure before without a reaction. Possible explanations of the death are: 1. Sensitivity to the drug had developed after the previous application. 2. The solution had been aspirated into the lungs. 3. The solution had become too concentrated; owing to evaporation from the container, it had been concentrated from 2 to 6 per cent.

HITSCHLER, Philadelphia.

LINGUISTIC PROBLEMS OF DEAFENED VETERANS RETURNING TO THE UNIVERSITIES. ALICE M. WELSH, Quart. J. Speech **32**:340, 1946.

One thousand three hundred and thirty deafened veterans enrolled at the University of Minnesota, beginning with the fall quarter of 1946. Speech clinics have the responsibility of locating these veterans, who tend to develop a policy of isolation. Although thousands of hearing losses were detected and treated in government hospitals, deficiencies of a progressive or permanent nature will be the responsibility of speech clinics for years to come. At the University of Minnesota an extensive counseling program has been established to enable veterans to readjust to civilian life with greater ease. The usual veteran is concerned with his deteriorating speech and loss of ability to comprehend. This handicaps him greatly in college activities. An aural rehabilitation program must provide classes in speech reading, facilities for the correction and the prevention of articulatory and phonatory deterioration, recommendations when the need for a hearing aid has been indicated, and physical and psychologic counseling concerning social, vocational and academic problems. Medical consultation is advisable. Often the audiogram alone does not reflect accurately or definitely the degree of difficulty the veteran will have in hearing or imitating speech. The hearing aid is the greatest single therapeutic aid, but speech training is usually necessary even after the aid has been fitted. The small amount of training given in lip reading and

speech reading and in speech in the special government centers has made many veterans desire to continue this work. Most deafened veterans are eager to take advantage of all available services of the university. The speech pathologist must demonstrate to the dubious ones the need for objectivity and early rehabilitation.

PALMER, Wichita, Kan.

A SUMMARY OF THE FINDINGS IN TWO HUNDRED CONSECUTIVE OPERATED CASES OF NECK INFECTION OF DENTAL ORIGIN. ARTHUR M. ALDEN, South. M. J. **38**:327 (May) 1945.

Alden calls attention to the fact that until the last decade the mortality of patients with deep infections of the neck was high, the rate ranging in the neighborhood of 40 per cent. In more recent years the rate has been greatly reduced, owing to better training and greater skill of the men doing the work and to the advent of chemotherapeutic agents such as the sulfonamide compounds and penicillin. In locating the pus and draining the abscess cavity a thorough knowledge of the ramifications of the cervical fascia is essential.

In this series of cases pus was limited to the submental space in 21 cases, to the submental and submaxillary space in 37 cases, to the submaxillary space in 74 cases, to the submaxillary and parapharyngeal spaces in 29 cases, to the parapharyngeal space in 22 cases, to the subparotid space in 13 cases and to the pterygomaxillary space in 4 cases. The submaxillary space was involved alone or in association with other spaces in 68 per cent of all cases.

Cultures showed a preponderance of Vincent's organisms and Bacillus fusiformis; these organisms were often associated with streptococci or staphylococci or both. Trismus is a frequent symptom in these cases; it is due to irritation of the internal pterygoid muscle of the affected side and occurs when there is infection of the parapharyngeal space. Osteomyelitis and edema of the larynx are not infrequent complications in these cases. Early tracheotomy is imperative in the presence of laryngeal edema.

The patients should be hospitalized immediately; if they are dehydrated, intravenous and intranasal administration of fluids is indicated. Neoarsphenamine is given in doses of 0.3 to 0.5 Gm., depending on the weight and the condition of the patient. If streptococci are present, sulfadiazine in amounts sufficient to establish and maintain a blood level of 6 to 7 mg. per hundred cubic centimeters is indicated. In severe involvement adequate doses of penicillin are indicated. These patients should be operated on as soon as the diffuse cellulitis becomes definitely localized. In cases of trismus the operation should be done with the region under local anesthesia, otherwise a respiratory fatality may occur. The type and the extent of the incision will depend on the location of the pus. After the cavity has been thoroughly opened and emptied, it is packed with dental rubber 2 inches (5 cm.) wide and kept open as long as necessary. A detailed case report of the only fatality of the entire series is given. The article is well worth reading.

SINGLETON, Dallas, Texas.

IS THE BIOPSY OF NEOPLASM DANGEROUS? M. E. MAUN and W. F. DUNNING, Surg., Gynec. & Obst. **82**:567 (May) 1946.

Recent reports appearing in the literature have indicated that removal or destruction of any quantity of a neoplastic growth prolongs the survival period of the host in proportion to the extent of the procedure. Using rats bearing transplanted fibrosarcoma, adenocarcinoma and squamous cell carcinoma, plus a careful control series, the authors found that the survival period of the animals was significantly prolonged by removal of a considerable mass of tumor. In the fibrosarcoma series the percentage with pulmonary and lymph node metastases was not altered by the surgical removal of neoplasm. In the adenocarcinoma series there was a higher rate of pulmonary metastasis in the surgically treated

group. Simple biopsy performed on rats with transplanted adenocarcinoma and squamous carcinoma did not affect the survival period or change the percentage having metastases in lymph nodes, lungs and skeleton. FRIEDBERG, Chicago.

EXTERNAL OPERATIONS FOR REMOVING FOREIGN BODIES FROM THE ESOPHAGUS. HARRY BJÖRK, Acta oto-laryng. **34**:131, 1946.

Björk calls attention to the endoscopic methods developed by laryngologists and to the fact that procedures by which foreign bodies may be removed from the esophagus under direct vision have gradually supplanted blind applications of probangs and coin catchers and external operations. But he also draws attention to the fact that laryngologists have become so obsessed with the idea that esophageal foreign bodies may be removed by endoscopic methods that they have lost sight of the fact that in certain cases it might be safer to have recourse to the external operation than to aggravate the situation by repeated peroral attempts at extraction. These repeated attempts to extract the foreign body with the esophagoscope frequently give rise to inflammatory complications which in not a few cases lead to death of the patient. He believes that although the endoscopic methods are the methods of choice they should go hand in hand with the external operation and that there will always be a place for the external operation in selected cases. He calls attention to the 1940 statistics of Huizinga, who stated that foreign bodies were removed by the external approach in 6 of 125 cases with no mortality.

Björk calls attention to five levels at which constriction of the esophagus may encourage the lodgment of foreign bodies, namely, that of the cricoid cartilage, that of the upper thoracic aperture, that of the aorta, that of the left main bronchus and that of the diaphragm. According to most authors, the majority of foreign bodies lodge at the upper two levels of constriction—in the postcricoid region and the region of the jugulum sterni. This fact is of great importance as the cervical portion of the esophagus lends itself best to the external approach.

The middle portion of the esophagus is most difficult to approach via an external route, but according to H. Killian it is possible to reach the part from 2 to 3 cm. below the bifurcation of the trachea by the cervical external operation, and according to Anschutz the lower part of the esophagus can be explored for a distance of 12 to 15 cm. above the cardia by way of a gastrotomy. Thoracic esophagotomy should, in Björk's opinion, be completely discarded because of its dangers.

Many different combinations of esophagoscopy and external operation are possible. Von Hacker's so-called trachelotomia lateralis is the most important one. The esophagus is exposed but not opened as in esophagotomy. The impacted foreign body is palpated and displaced by being manipulated through the intact esophageal wall. It is then extracted by means of the esophagoscope. The second combined method proposed by von Hacker is inferior esophagoscopy in which, after the usual cervical esophagotomy, the esophagoscope is introduced through the incision in the esophagus. The third combined intervention is retrograde esophagoscopy in connection with gastrotomy. Finally, in cases in which mediastinitis already exists Schlemmer has proposed that the foreign body be first removed by endoscopic methods, to be followed immediately by the external operation for purposes of drainage.

The size and the shape of the foreign body are of great importance. Dentures are particularly difficult to remove by means of the esophagoscope. In 3 of 4 cases published in Finland in which the external approach was used for extraction the foreign body was a denture.

Roentgen examination should always be resorted to in connection with those foreign bodies of the esophagus which are serious enough to warrant the external approach. Many foreign bodies are not radiopaque, and for these Björk advocates the method proposed by Schmidt in 1938, in which a thin barium sulfate mixture is swallowed. This has a double advantage. It coats the foreign body with the

barium compound and renders it opaque and if a perforation has already taken place the barium sulfate mixture flows into the fistula.

If, after endoscopic extraction of the foreign body, signs of inflammation are present in the mediastinum, such as pain, swelling, increase of temperature or emphysema, a rapid external intervention is indicated. As to the external operation itself, it should be done on the left side unless the emphysema and inflammatory symptoms are exclusively on the right side, because the cervical portion of the esophagus normally curves to the left. Incision is made along the anterior margin of the sternocleidomastoid muscle, and one picks one's way carefully between the great vessel column and the thyroid gland, taking care not to injure the inferior thyroid artery. The foreign body usually causes a bulge in the wall of the esophagus or is made to do so when the patient dilates the esophagus in the act of swallowing. The recurrent laryngeal nerve runs along the lateral surface of the esophagus and can easily be injured if the esophagus is carelessly exposed or opened. The esophageal incision is made lengthwise, and after the removal of the foreign body the incision is closed with catgut. The operative wound is left open and drained.

Björk then describes 3 cases in which he removed a foreign body by cervical esophagotomy. All 3 patients recovered. Antibiotics were freely used.

GROVE, Milwaukee.

Directory of Otolaryngologic Societies *

INTERNATIONAL

First Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Secretary: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit, Mich.
Place: Chicago. Time: June 21-25, 1948.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
President-Elect: Dr. C. H. McCaskey, 20 N. Meridian St., Indianapolis, Ind.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 12-17, 1947.

American Broncho-Esophagological Association

President: Dr. Millard F. Arbuckle, 539 N. Grand Blvd., St. Louis.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11.

American Laryngological Association

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2.
Place: Old Homestead, Va. Time: April 14-15, 1948.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Lyman G. Richards, 15 Whiting Rd., Wellesley Hills, Mass.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Chalfonte-Haddon Hall, Atlantic City. Time: April 7-9, 1948.

SECTIONS:

Eastern.—Chairman: Dr. Robert F. Ridpath, 18th and Chestnut Sts., Philadelphia.
Place: New York. Time: Jan. 16, 1948.
Southern.—Chairman: Dr. Walter T. Hotchkiss, 541 Lincoln Rd., Miami Beach, Fla.
Place: New Orleans. Time: Jan. 23, 1948.
Middle.—Chairman: Dr. Oliver E. Van Alyea, 135 S. La Salle St., Chicago.
Place: Columbus, Ohio. Time: Jan. 19, 1948.
Western.—Chairman: Dr. Colby Hall, 1136 W. 6th St., Los Angeles, Calif.
Place: San Francisco. Time: Jan. 31-Feb. 1, 1948.

American Otological Society

President: Dr. Bernard J. McMahon, 634 N. Grand Blvd., St. Louis.
Secretary: Dr. Gordon D. Hoople, 713 E. Genesee St., Syracuse, N. Y.
Place: Old Homestead, Va. Time: April 12-13, 1948.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Alfred Schattner, 115 E. 61st St., New York.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven, Conn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.